Outstanding men in a number of fields contributed their brilliant and original ideas to the now-famous Hixon Symposium. Here is the skillfully edited record of that Symposium.

Edited by Lloyd A. Jeffress

Lloyd A. Jeffress
Henry W. Brosin
Ward C. Halstead
Heinrich Klüver
Wolfgang Köhler
K. S. Lashley
Warren S. McCulloch
John von Neumann
Ralph W. Gerard
H. S. Liddell
Donald B. Lindsley
R. Lorente de Nó
J. M. Nielsen
Paul Weiss
A. van Harreveld
Linus Pauling
John Stroud
C. A. G. Wiersma
Lowell Woodbury

CEREBRAL MECHANISMS in BEHAVIOR

The Hixon Symposium

ut the Hixon Symposium and
record of its proceedings—

rom September 20-25, 1948, sev-
men met at the California Institute
Technology to take part in the Hixon
nposium. Some contributed papers,
ne were on a discussion panel, some
gmented the discussion by brief re-
rks—all were widely known leaders
their fields. The general subject of
symposium was "Cerebral Mech-
isms in Behavior," and in contribut-
g their stimulating and original ideas,
e members struck at the periphery of
number of fields, successfully break-
g down the barriers among many
isciplines. In this way, the ideas ex-
ressed in the Hixon Symposium bear
great resemblance to those that form
he study of cybernetics, and should
nterest all those who were concerned
with Professor Wiener's important
ook.

This written record of the Symposium consists of the six major papers, each followed by a discussion section, with two additional discussion sections and a final evaluation also included. About half the space in the book is devoted to the formal papers and about half to informal discussion.

Psychiatrists, physiologists, neurologists, neuropsychiatrists, psychologists, and anyone in a related field, will find this carefully compiled book of immense value and interest.

G-MJP-750

7.50

1024
16.7.56

150.1943
JEE

A019413
J.46.

CEREBRAL MECHANISMS IN BEHAVIOR

The Hixon Symposium

CEREBRAL MECHANISMS IN BEHAVIOR

The Hixon Symposium

Edited by

LLOYD A. JEFFRESS

PROFESSOR OF PSYCHOLOGY
UNIVERSITY OF TEXAS

Bureau Ednl. Psy. Research
DAVID HA [illegible] ING COLLEGE
Dated 16. 3. 56
Accs. No 1024

NEW YORK · JOHN WILEY & SONS, INC.
LONDON · CHAPMAN & HALL, LIMITED

COPYRIGHT, 1951
BY
JOHN WILEY & SONS, INC.

All Rights Reserved
This book or any part thereof must not be reproduced in any form without the written permission of the publisher.

PRINTED IN THE UNITED STATES OF AMERICA

Members of the Hixon Symposium

GENERAL CHAIRMAN

LLOYD A. JEFFRESS, Professor of Psychology, University of Texas (Hixon Visiting Professor, California Institute of Technology, 1947–1948)

CHAIRMAN OF THE MEETINGS

HENRY W. BROSIN, Professor of Psychiatry, University of Pittsburgh, and Director, Western Psychiatric Institute and Clinic

PAPERS

WARD C. HALSTEAD, Professor of Experimental Psychology, University of Chicago, Departments of Medicine and Psychology

HEINRICH KLÜVER, Professor of Experimental Psychology, Division of the Biological Sciences, University of Chicago

WOLFGANG KÖHLER, Research Professor of Philosophy and Psychology, Swarthmore College

K. S. LASHLEY, Research Professor of Neuropsychology, Harvard University, and Director, Yerkes Laboratories of Primate Biology

WARREN S. MCCULLOCH, Professor of Psychiatry, University of Illinois College of Medicine, and Illinois Neuropsychiatric Institute

JOHN VON NEUMANN, Professor of Mathematics, Institute for Advanced Study

DISCUSSION PANEL

RALPH W. GERARD, Professor of Physiology, University of Chicago

H. S. LIDDELL, Professor of Psychobiology, Cornell University

DONALD B. LINDSLEY, Professor of Psychology, Northwestern University

R. LORENTE DE NÓ, Member of the Rockefeller Institute for Medical Research

J. M. NIELSEN, Clinical Professor of Neurology and Psychiatry, University of Southern California

PAUL WEISS, Professor of Zoology, University of Chicago

ADDITIONAL PARTICIPANTS

A. van Harreveld, Professor of Physiology, California Institute of Technology

Linus Pauling, Professor of Chemistry, California Institute of Technology

John Stroud, U.S. Navy Electronics Laboratory

C. A. G. Wiersma, Professor of Physiology, California Institute of Technology

Lowell Woodbury, Assistant Professor of Physiology, University of Utah Medical School

Preface

This book is the report of a symposium held at the California Institute of Technology in September, 1948. The topic of the symposium was "Cerebral Mechanisms in Behavior." To discuss it a group was brought to the Institute for the week of September 20 to September 25 under the auspices of the Hixon Fund Committee.

The Hixon Fund was established in 1938 by a grant to the California Institute of Technology from the estate of Frank P. Hixon. Its purpose has been to support scientific endeavor which offers promise of increased understanding of human behavior. Among the projects supported by the Hixon Fund have been the following:

A period of collaboration between Dr. Lorente de Nó of the Rockefeller Institute for Medical Research and Dr. Leverett Davis of the Physics Department of the California Institute of Technology upon the distribution of electrical potential in nerves.

A cooperative study by members of the Institute staff under the leadership of Dr. Wiersma and Dr. van Harreveld, and representatives of the California Department of Institutions, dealing with electroconvulsive therapy and with electronarcosis as a therapeutic agent in mental disorders.

A study by Dr. David B. Tyler of treatments for cases of battle fatigue and of motion sickness.

A study by Dr. Marjory Douglis of social behavior in chickens.

The Hixon Fund is at present administered by a committee consisting of Professors G. W. Beadle, James Bonner, Henry Borsook, Max Delbrück, Linus Pauling, A. H. Sturtevant, C. A. G. Wiersma, and A. van Harreveld, chairman. Dr. Max Mason, who had been chairman of the committee ever since the establishment of the Hixon Fund, has recently retired from it.

In 1947 the Committee invited me to the Institute to spend the year in research in audition and to help them in organizing the 1948 symposium. The year, which was a very pleasant one for me, culminated in the exciting symposium week.

I wish it were possible to convey to the reader more of the atmosphere of the meetings than can be done in type. Smiles, frowns, gestures, and head shakings often add meaning to verbal discussion, but do not print well. The many compliments paid by the speakers to one another contributed to the friendliness of the occasion but could not be preserved.

Much credit for the liveliness of the meetings goes to Hank Brosin. He was chairman of the first session. According to the program, other members of the group were to be chairmen of later sessions, but he conducted his meeting with such brilliance that the group unanimously decided to throw the program overboard and elect him permanent chairman. He thoroughly justified the decision. He introduced the speakers with gems of biography, he kept the discussion under control, called on people for comments (thereby keeping them alert), and altogether charmed everyone with his execution. It is one of my chief regrets that most of this had to be sacrificed to the exigencies of publication.

Stenotype operators recorded the symposium talks and the discussions. The records were then transcribed, mimeographed, and distributed to the speakers for correction. The revised discussion sections constituted the raw material for my work as editor. This material had to be cut and patched together; repetitious sections, unanswered questions, and other fragments were omitted. The major papers were left as written. Galley proof was sent to all participants for final correction (and, it turned out, further revision). This procedure has taken time, but the result is probably a better book, and one more nearly representing the authors' views, than could have been brought out much sooner.

I am sorry that I am not able to include Dr. Lorente de Nó's paper in the book. Since much of his material was presented in the form of slides, the stenotype notes are incomplete, and his ill health during the last two years has made it impossible for him to provide a manuscript of his paper or of his discussion remarks. Accordingly I must take the responsibility for any inaccuracies or misstatements in the comments attributed to him.

I am happy to acknowledge indebtedness to many people—to the participants in the symposium for their help with the manuscript and the proof, to the University of Chicago Press for permission to quote from Professor Breasted's book *The Edwin Smith Surgical Papyrus*, to Hans Huber for permission to quote from Hermann Rorschach's *Psychodiagnostics*, and to the publishers mentioned in the text for permission to reproduce many of the illustrations. I am especially indebted to my wife, Sylvia Jeffress, for her help with the manuscript and for her careful reading of the galley and page proofs.

Finally I wish to thank the Hixon Committee and the California Institute of Technology for making the symposium possible, and for giving me the opportunity to help.

LLOYD A. JEFFRESS

Austin, Texas
September, 1951

Contents

THE GENERAL AND LOGICAL THEORY OF AUTOMATA 1

by JOHN VON NEUMANN

Discussion 32

WHY THE MIND IS IN THE HEAD 42

by WARREN S. MCCULLOCH

Discussion 57

GENERAL DISCUSSION 82

THE PROBLEM OF SERIAL ORDER IN BEHAVIOR 112

by K. S. LASHLEY

Discussion 136

FUNCTIONAL DIFFERENCES BETWEEN THE OCCIPITAL AND TEMPORAL LOBES WITH SPECIAL REFERENCE TO THE INTERRELATIONS OF BEHAVIOR AND EXTRACEREBRAL MECHANISMS 147

by HEINRICH KLÜVER

Discussion 182

RELATIONAL DETERMINATION IN PERCEPTION 200

by WOLFGANG KÖHLER

Discussion 230

BRAIN AND INTELLIGENCE 244

by WARD C. HALSTEAD

Discussion 272

GENERAL DISCUSSION 279

THE SYMPOSIUM FROM THE VIEWPOINT OF A CLINICIAN 289
by Henry W. Brosin

NAME INDEX 301

SUBJECT INDEX 305

The General and Logical Theory of Automata[*]

JOHN VON NEUMANN
The Institute for Advanced Study

I have to ask your forbearance for appearing here, since I am an outsider to most of the fields which form the subject of this conference. Even in the area in which I have some experience, that of the logics and structure of automata, my connections are almost entirely on one side, the mathematical side. The usefulness of what I am going to say, if any, will therefore be limited to this: I may be able to give you a picture of the mathematical approach to these problems, and to prepare you for the experiences that you will have when you come into closer contact with mathematicians. This should orient you as to the ideas and the attitudes which you may then expect to encounter. I hope to get your judgment of the modus procedendi and the distribution of emphases that I am going to use. I feel that I need instruction even in the limiting area between our fields more than you do, and I hope that I shall receive it from your criticisms.

Automata have been playing a continuously increasing, and have by now attained a very considerable, role in the natural sciences. This is a process that has been going on for several decades. During the last part of this period automata have begun to invade certain parts of mathematics too—particularly, but not exclusively, mathematical physics or applied mathematics. Their role in mathematics presents an interesting counterpart to certain functional aspects of organization in nature. Natural organisms are, as a rule, much more complicated and subtle, and therefore much less well understood in detail, than are artificial automata. Nevertheless, some regularities which we observe in the organization of the former may be quite instructive in our thinking and planning of the latter; and conversely, a good deal of our experiences and difficulties with our artificial automata can be to some extent projected on our interpretations of natural organisms.

---

[*] This paper is an only slightly edited version of one that was read at the Hixon Symposium on September 20, 1948, in Pasadena, California. Since it was delivered as a single lecture, it was not feasible to go into as much detail on every point as would have been desirable for a final publication. In the present write-up it seemed appropriate to follow the dispositions of the talk; therefore this paper, too, is in many places more sketchy than desirable. It is to be taken only as a general outline of ideas and of tendencies. A detailed account will be published on another occasion.

PRELIMINARY CONSIDERATIONS

Dichotomy of the Problem: Nature of the Elements, Axiomatic Discussion of Their Synthesis. In comparing living organisms, and, in particular, that most complicated organism, the human central nervous system, with artificial automata, the following limitation should be kept in mind. The natural systems are of enormous complexity, and it is clearly necessary to subdivide the problem that they represent into several parts. One method of subdivision, which is particularly significant in the present context, is this: The organisms can be viewed as made up of parts which to a certain extent are independent, elementary units. We may, therefore, to this extent, view as the first part of the problem the structure and functioning of such elementary units individually. The second part of the problem consists of understanding how these elements are organized into a whole, and how the functioning of the whole is expressed in terms of these elements.

The first part of the problem is at present the dominant one in physiology. It is closely connected with the most difficult chapters of organic chemistry and of physical chemistry, and may in due course be greatly helped by quantum mechanics. I have little qualification to talk about it, and it is not this part with which I shall concern myself here.

The second part, on the other hand, is the one which is likely to attract those of us who have the background and the tastes of a mathematician or a logician. With this attitude, we will be inclined to remove the first part of the problem by the process of axiomatization, and concentrate on the second one.

The Axiomatic Procedure. Axiomatizing the behavior of the elements means this: We assume that the elements have certain well-defined, outside, functional characteristics; that is, they are to be treated as "black boxes." They are viewed as automatisms, the inner structure of which need not be disclosed, but which are assumed to react to certain unambiguously defined stimuli, by certain unambiguously defined responses.

This being understood, we may then investigate the larger organisms that can be built up from these elements, their structure, their functioning, the connections between the elements, and the general theoretical regularities that may be detectable in the complex syntheses of the organisms in question.

I need not emphasize the limitations of this procedure. Investigations of this type may furnish evidence that the system of axioms used is convenient and, at least in its effects, similar to reality. They are, however, not the ideal method, and possibly not even a very effective method, to determine the validity of the axioms. Such determinations of validity belong primarily to the first part of the problem. Indeed they are essentially covered by the properly physiological (or chemical or physical-chemical) determinations of the nature and properties of the elements.

The Significant Orders of Magnitude. In spite of these limitations, however, the "second part" as circumscribed above is important and difficult. With any reasonable definition of what constitutes an element, the natural organisms are very highly complex aggregations of these elements. The number of cells in the human body is somewhere of the general order of 10^{15} or 10^{16}. The number of neurons in the central nervous system is somewhere of the order of 10^{10}. We have absolutely no past experience with systems of this degree of complexity. All artificial automata made by man have numbers of parts which by any comparably schematic count are of the order 10^3 to 10^6. In addition, those artificial systems which function with that type of logical flexibility and autonomy that we find in the natural organisms do not lie at the peak of this scale. The prototypes for these systems are the modern computing machines, and here a reasonable definition of what constitutes an element will lead to counts of a few times 10^3 or 10^4 elements.

DISCUSSION OF CERTAIN RELEVANT TRAITS OF COMPUTING MACHINES

Computing Machines—Typical Operations. Having made these general remarks, let me now be more definite, and turn to that part of the subject about which I shall talk in specific and technical detail. As I have indicated, it is concerned with artificial automata and more specially with computing machines. They have some similarity to the central nervous system, or at least to a certain segment of the system's functions. They are of course vastly less complicated, that is, smaller in the sense which really matters. It is nevertheless of a certain interest to analyze the problem of organisms and organization from the point of view of these relatively small, artificial automata, and to effect their comparisons with the central nervous system from this frog's-view perspective.

I shall begin by some statements about computing machines as such.

The notion of using an automaton for the purpose of computing is relatively new. While computing automata are not the most complicated artificial automata from the point of view of the end results they achieve, they do nevertheless represent the highest degree of complexity in the sense that they produce the longest chains of events determining and following each other.

There exists at the present time a reasonably well-defined set of ideas about when it is reasonable to use a fast computing machine, and when it is not. The criterion is usually expressed in terms of the multiplications involved in the mathematical problem. The use of a fast computing machine is believed to be by and large justified when the computing task involves about a million multiplications or more in a sequence.

An expression in more fundamentally logical terms is this: In the relevant fields (that is, in those parts of [usually applied] mathematics, where the use of such machines is proper) mathematical experience indicates the desirability of precisions of about ten decimal places. A single multiplication would therefore seem to involve at least 10 × 10 steps (digital multiplications); hence a million multiplications amount to at least 10^8 operations. Actually, however, multiplying two decimal digits is not an elementary operation. There are various ways of breaking it down into such, and all of them have about the same degree of complexity. The simplest way to estimate this degree of complexity is, instead of counting decimal places, to count the number of places that would be required for the same precision in the binary system of notation (base 2 instead of base 10). A decimal digit corresponds to about three binary digits, hence ten decimals to about thirty binary. The multiplication referred to above, therefore, consists not of 10 × 10, but of 30 × 30 elementary steps, that is, not 10^2, but 10^3 steps. (Binary digits are "all or none" affairs, capable of the values 0 and 1 only. Their multiplication is, therefore, indeed an elementary operation. By the way, the equivalent of 10 decimals is 33 [rather than 30] binaries—but 33 × 33, too, is approximately 10^3.) It follows, therefore, that a million multiplications in the sense indicated above are more reasonably described as corresponding to 10^9 elementary operations.

Precision and Reliability Requirements. I am not aware of any other field of human effort where the result really depends on a sequence of a billion (10^9) steps in any artifact, and where, furthermore, it has the characteristic that every step actually matters—or, at least, may matter with a considerable probability. Yet, precisely this is true for computing machines—this is their most specific and most difficult characteristic.

Indeed, there have been in the last two decades automata which did perform hundreds of millions, or even billions, of steps before they produced a result. However, the operation of these automata is not serial. The large number of steps is due to the fact that, for a variety of reasons, it is desirable to do the same experiment over and over again. Such cumulative, repetitive procedures may, for instance, increase the size of the result, that is (and this is the important consideration), increase the significant result, the "signal," relative to the "noise" which contaminates it. Thus any reasonable count of the number of reactions which a microphone gives before a verbally interpretable acoustic signal is produced is in the high tens of thousands. Similar estimates in television will give tens of millions, and in radar possibly many billions. If, however, any of these automata makes mistakes, the mistakes usually matter only to the extent of the fraction of the total number of steps which they represent. (This is not exactly true in all relevant examples, but it represents the qualitative situation better than the opposite statement.) Thus the larger the number of operations required to produce a result, the smaller will be the significant contribution of every individual operation.

In a computing machine no such rule holds. Any step is (or may potentially be) as important as the whole result; any error can vitiate the result in its entirety. (This statement is not absolutely true, but probably nearly 30 per cent of all steps made are usually of this sort.) Thus a computing machine is one of the exceptional artifacts. They not only have to perform a billion or more steps in a short time, but in a considerable part of the procedure (and this is a part that is rigorously specified in advance) they are permitted not a single error. In fact, in order to be sure that the whole machine is operative, and that no potentially degenerative malfunctions have set in, the present practice usually requires that no error should occur anywhere in the entire procedure.

This requirement puts the large, high-complexity computing machines in an altogether new light. It makes in particular a comparison between the computing machines and the operation of the natural organisms not entirely out of proportion.

The Analogy Principle. All computing automata fall into two great classes in a way which is immediately obvious and which, as you will see in a moment, carries over to living organisms. This classification is into analogy and digital machines.

Let us consider the analogy principle first. A computing machine may be based on the principle that numbers are represented by certain physical quantities. As such quantities we might, for instance, use the intensity of an electrical current, or the size of an electrical potential, or the number of degrees of arc by which a disk has been rotated (possibly in conjunction with the number of entire revolutions effected), etc. Operations like addition, multiplication, and integration may then be performed by finding various natural processes which act on these quantities in the desired way. Currents may be multiplied by feeding them into the two magnets of a dynamometer, thus producing a rotation. This rotation may then be transformed into an electrical resistance by the attachment of a rheostat; and, finally, the resistance can be transformed into a current by connecting it to two sources of fixed (and different) electrical potentials. The entire aggregate is thus a "black box" into which two currents are fed and which produces a current equal to their product. You are certainly familiar with many other ways in which a wide variety of natural processes can be used to perform this and many other mathematical operations.

The first well-integrated, large computing machine ever made was an analogy machine, V. Bush's Differential Analyzer. This machine, by the way, did the computing not with electrical currents, but with rotating disks. I shall not discuss the ingenious tricks by which the angles of rotation of these disks were combined according to various operations of mathematics.

I shall make no attempt to enumerate, classify, or systematize the wide variety of analogy principles and mechanisms that can be used in computing. They are confusingly multiple. The guiding principle without which it is impossible to reach an understanding of the situation is the classical one of all "communication theory"—the "signal to noise ratio." That is, the critical question with every analogy procedure is this: How large are the uncontrollable fluctuations of the mechanism that constitute the "noise," compared to the significant "signals" that express the numbers on which the machine operates? The usefulness of any analogy principle depends on how low it can keep the relative size of the uncontrollable fluctuations—the "noise level."

To put this in another way. No analogy machine exists which will really form the product of two numbers. What it will form is this product, plus a small but unknown quantity which represents the random noise of the mechanism and the physical processes involved. The whole problem is to keep this quantity down. This principle has controlled the entire relevant technology. It has, for instance, caused the adoption of seemingly complicated and clumsy mechanical devices instead of the simpler and elegant electrical ones. (This, at least, has been the case throughout most of the last twenty years. More recently, in certain applications which required only very limited precision the electrical devices have again come to the fore.) In comparing mechanical with electrical analogy processes, this roughly is true: Mechanical arrangements may bring this noise level below the "maximum signal level" by a factor of something like $1{:}10^4$ or 10^5. In electrical arrangements, the ratio is rarely much better than $1{:}10^2$. These ratios represent, of course, errors in the elementary steps of the calculation, and not in its final results. The latter will clearly be substantially larger.

The Digital Principle. A digital machine works with the familiar method of representing numbers as aggregates of digits. This is, by the way, the procedure which all of us use in our individual, non-mechanical computing, where we express numbers in the decimal system. Strictly speaking, digital computing need not be decimal. Any integer larger than one may be used as the basis of a digital notation for numbers. The decimal system (base 10) is the most common one, and all digital machines built to date operate in this system. It seems likely, however, that the binary (base 2) system will, in the end, prove preferable, and a number of digital machines using that system are now under construction.

The basic operations in a digital machine are usually the four species of arithmetic: addition, subtraction, multiplication, and division. We might at first think that, in using these, a digital machine possesses (in contrast to the analogy machines referred to above) absolute precision. This, however, is not the case, as the following consideration shows.

Take the case of multiplication. A digital machine multiplying two 10-digit numbers will produce a 20-digit number, which is their product, with no error whatever. To this extent its precision is absolute, even though the electrical or mechanical components of the arithmetical organ of the machine are as such of limited precision. As long as there is no breakdown of some component, that is, as long as the operation of each component produces only fluctuations within its preassigned tolerance limits, the result will be absolutely correct. This is, of course, the great and characteristic virtue of the digital procedure. Error, as a matter of normal operation and not solely (as indicated above) as an accident attributable to some definite breakdown, nevertheless creeps in, in the following manner. The absolutely correct product of two 10-digit numbers is a 20-digit number. If the machine is built to handle 10-digit numbers only, it will have to disregard the last 10 digits of this 20-digit number and work with the first 10 digits alone. (The small, though highly practical, improvement due to a possible modification of these digits by "round-off" may be disregarded here.) If, on the other hand, the machine can handle 20-digit numbers, then the multiplication of two such will produce 40 digits, and these again have to be cut down to 20, etc., etc. (To conclude, no matter what the maximum number of digits is for which the machine has been built, in the course of successive multiplications this maximum will be reached, sooner or later. Once it has been reached, the next multiplication will produce supernumerary digits, and the product will have to be cut to half of its digits [the first half, suitably rounded off]. The situation for a maximum of 10 digits is therefore typical, and we might as well use it to exemplify things.)

Thus the necessity of rounding off an (exact) 20-digit product to the regulation (maximum) number of 10 digits introduces in a digital machine qualitatively the same situation as was found above in an analogy machine. What it produces when a product is called for is not that product itself, but rather the product plus a small extra term—the round-off error. This error is, of course, not a random variable like the noise in an analogy machine. It is, arithmetically, completely determined in every particular instance. Yet its mode of determination is so complicated, and its variations throughout the number of instances of its occurrence in a problem so irregular, that it usually can be treated to a high degree of approximation as a random variable.

(These considerations apply to multiplication. For division the situation is even slightly worse, since a quotient can, in general, not be expressed with absolute precision by any finite number of digits. Hence here rounding off is usually already a necessity after the first operation. For addition and subtraction, on the other hand, this difficulty does not arise: The sum or difference has the same number of digits [if there is no increase in size beyond the planned maximum] as the addends themselves. Size may create difficulties which are added to the difficulties of precision discussed here, but I shall not go into these at this time.)

The Role of the Digital Procedure in Reducing the Noise Level. The important difference between the noise level of a digital machine, as described above, and of an analogy machine is not qualitative at all; it is quantitative. As pointed out above, the relative noise level of an analogy machine is never lower than 1 in 10^5, and in many cases as high as 1 in 10^2. In the 10-place decimal digital machine referred to above the relative noise level (due to round-off) is 1 part in 10^{10}. Thus the real importance of the digital procedure lies in its ability to reduce the computational noise level to an extent which is completely unobtainable by any other (analogy) procedure. In addition, further reduction of the noise level is increasingly difficult in an analogy mechanism, and increasingly easy in a digital one. In an analogy machine a precision of 1 in 10^3 is easy to achieve; 1 in 10^4 somewhat difficult; 1 in 10^5 very difficult; and 1 in 10^6 impossible, in the present state of technology. In a digital machine, the above precisions mean merely that one builds the machine to 3, 4, 5, and 6 decimal places, respectively. Here the transition from each stage to the next one gets actually easier. Increasing a 3-place machine (if anyone wished to build such a machine) to a 4-place machine is a 33 per cent increase; going from 4 to 5 places, a 20 per cent increase; going from 5 to 6 places, a 17 per cent increase. Going from 10 to 11 places is only a 10 per cent increase. This is clearly an entirely different milieu, from the point of view of the reduction of "random noise," from that of physical processes. It is here—and not in its practically ineffective absolute reliability—that the importance of the digital procedure lies.

COMPARISONS BETWEEN COMPUTING MACHINES AND LIVING ORGANISMS

Mixed Character of Living Organisms. When the central nervous system is examined, elements of both procedures, digital and analogy, are discernible.

The neuron transmits an impulse. This appears to be its primary function, even if the last word about this function and its exclusive or non-exclusive character is far from having been said. The nerve impulse seems in the main to be an all-or-none affair, comparable to a binary digit. Thus a digital element is evidently present, but it is equally evident that this is not the entire story. A great deal of what goes on in the organism is not mediated in this manner, but is dependent on the general chemical composition of the blood stream or of other humoral media. It is well known that there are various composite functional sequences in the organism which have to go through a variety of steps from the original stimulus to the ultimate effect—some of the steps being neural, that is, digital, and others humoral, that is, analogy. These digital and analogy portions in such a chain may alternately multiply. In certain cases of this type, the chain can actually feed back into itself, that is, its ultimate output may again stimulate its original input.

It is well known that such mixed (part neural and part humoral) feedback chains can produce processes of great importance. Thus the mechanism which keeps the blood pressure constant is of this mixed type. The nerve which senses and reports the blood pressure does it by a sequence of neural impulses, that is, in a digital manner. The muscular contraction which this impulse system induces may still be described as a superposition of many digital impulses. The influence of such a contraction on the blood stream is, however, hydrodynamical, and hence analogy. The reaction of the pressure thus produced back on the nerve which reports the pressure closes the circular feedback, and at this point the analogy procedure again goes over into a digital one. The comparisons between the living organisms and the computing machines are, therefore, certainly imperfect at this point. The living organisms are very complex—part digital and part analogy mechanisms. The computing machines, at least in their recent forms to which I am referring in this discussion, are purely digital. Thus I must ask you to accept this oversimplification of the system. Although I am well aware of the analogy component in living organisms, and it would be absurd to deny its importance, I shall, nevertheless, for the sake of the simpler discussion, disregard that part. I shall consider the living organisms as if they were purely digital automata.

Mixed Character of Each Element. In addition to this, one may argue that even the neuron is not exactly a digital organ. This point has been put forward repeatedly and with great force. There is certainly a great deal of truth in it, when one considers things in considerable detail. The relevant assertion is, in this respect, that the fully developed nervous impulse, to which all-or-none character can be attributed, is not an elementary phenomenon, but is highly complex. It is a degenerate state of the complicated electrochemical complex which constitutes the neuron, and which in its fully analyzed functioning must be viewed as an analogy machine. Indeed, it is possible to stimulate the neuron in such a way that the breakdown that releases the nervous stimulus will not occur. In this area of "subliminal stimulation," we find first (that is, for the weakest stimulations) responses which are proportional to the stimulus, and then (at higher, but still subliminal, levels of stimulation) responses which depend on more complicated non-linear laws, but are nevertheless continuously variable and not of the breakdown type. There are also other complex phenomena within and without the subliminal range: fatigue, summation, certain forms of self-oscillation, etc.

In spite of the truth of these observations, it should be remembered that they may represent an improperly rigid critique of the concept of an all-or-none organ. The electromechanical relay, or the vacuum tube, when properly used, are undoubtedly all-or-none organs. Indeed, they are the prototypes of such organs. Yet both of them are in reality complicated analogy mechanisms, which upon appropriately adjusted stimulation respond continuously, linearly or non-linearly, and exhibit the phenomena of "breakdown" or "all-or-none" response only under very particular conditions of operation. There is little difference between this performance and the above-described performance of neurons. To put it somewhat differently. None of these is an exclusively all-or-none organ (there is little in our technological or physiological experience to indicate that absolute all-or-none organs exist); this, however, is irrelevant. By an all-or-none organ we should rather mean one which fulfills the following two conditions. First, it functions in the all-or-none manner under certain suitable operating conditions. Second, these operating conditions are the ones under which it is normally used; they represent the functionally normal state of affairs within the large organism, of which it forms a part. Thus the important fact is not whether an organ has necessarily and under all conditions the all-or-none character—this is probably never the case—but rather whether in its proper context it functions primarily, and appears to be intended to function primarily, as an all-or-none organ. I realize that this definition brings in rather undesirable criteria of "propriety" of context, of "appearance" and "intention." I do not see, however, how we can avoid using them, and how we can forego counting on the employment of common sense in their application. I shall, accordingly, in what follows use the working hypothesis that the neuron is an all-or-none digital organ. I realize that the last word about this has not been said, but I hope that the above excursus on the limitations of this working hypothesis and the reasons for its use will reassure you. I merely want to simplify my discussion; I am not trying to prejudge any essential open question.

In the same sense, I think that it is permissible to discuss the neurons as electrical organs. The stimulation of a neuron, the development and progress of its impulse, and the stimulating effects of the impulse at a synapse can all be described electrically. The concomitant chemical and other processes are important in order to understand the internal functioning of a nerve cell. They may even be more important than the electrical phenomena. They seem, however, to be hardly necessary for a description of a neuron as a "black box," an organ of the all-or-none type. Again the situation is no worse here than it is for, say, a vacuum tube. Here, too, the purely electrical phenomena are accompanied by numerous other phenomena of solid state physics, thermodynamics, mechanics. All of these are important to understand the structure of a vacuum tube, but are best excluded from the discussion, if it is to treat the vacuum tube as a "black box" with a schematic description.

The Concept of a Switching Organ or Relay Organ. The neuron, as well as the vacuum tube, viewed under the aspects discussed above, are then two instances of the same generic entity, which it is customary to call a "switching organ" or "relay organ." (The electromechanical relay is, of course, another instance.) Such an organ is defined as a "black box," which responds to a specified stimulus or combination of stimuli by an energetically independent response. That is, the response is expected to have enough energy to cause several stimuli of the same kind as the ones which initiated it. The energy of the response, therefore, cannot have been supplied by the original stimulus. It must originate in a different and independent source of power. The stimulus merely directs, controls the flow of energy from this source.

(This source, in the case of the neuron, is the general metabolism of the neuron. In the case of a vacuum tube, it is the power which maintains the cathode-plate potential difference, irrespective of whether the tube is conducting or not, and to a lesser extent the heater power which keeps "boiling" electrons out of the cathode. In the case of the electromechanical relay, it is the current supply whose path the relay is closing or opening.)

The basic switching organs of the living organisms, at least to the extent to which we are considering them here, are the neurons. The basic switching organs of the recent types of computing machines are vacuum tubes; in older ones they were wholly or partially electromechanical relays. It is quite possible that computing machines will not always be primarily aggregates of switching organs, but such a development is as yet quite far in the future. A development which may lie much closer is that the vacuum tubes may be displaced from their role of switching organs in computing machines. This, too, however, will probably not take place for a few years yet. I shall, therefore, discuss computing machines solely from the point of view of aggregates of switching organs which are vacuum tubes.

Comparison of the Sizes of Large Computing Machines and Living Organisms. Two well-known, very large vacuum tube computing machines are in existence and in operation. Both consist of about 20,000 switching organs. One is a pure vacuum tube machine. (It belongs to the U. S. Army Ordnance Department, Ballistic Research Laboratories, Aberdeen, Maryland, designation "ENIAC.") The other is mixed—part vacuum tube and part electromechanical relays. (It belongs to the I. B. M. Corporation, and is located in New York, designation "SSEC.") These machines are a good deal larger than what is likely to be the size of the vacuum tube computing machines which will come into existence and operation in the next few years. It is probable that each one of these will consist of 2000 to 6000 switching organs. (The reason for this decrease lies in a different attitude about the treatment of the "memory," which I will not discuss here.) It is possible that in later years the machine sizes will increase again, but it is not likely that 10,000 (or perhaps a few times 10,000) switching organs will be exceeded as long as the present techniques and philosophy are employed. To sum up, about 10^4 switching organs seem to be the proper order of magnitude for a computing machine.

In contrast to this, the number of neurons in the central nervous system has been variously estimated as something of the order of 10^{10}. I do not know how good this figure is, but presumably the exponent at least is not too high, and not too low by more than a unit. Thus it is very conspicuous that the central nervous system is at least a million times larger than the largest artificial automaton that we can talk about at present. It is quite interesting to inquire why this should be so and what questions of principle are involved. It seems to me that a few very clear-cut questions of principle are indeed involved.

Determination of the Significant Ratio of Sizes for the Elements. Obviously, the vacuum tube, as we know it, is gigantic compared to a nerve cell. Its physical volume is about a billion times larger, and its energy dissipation is about a billion times greater. (It is, of course, impossible to give such figures with a unique validity, but the above ones are typical.) There is, on the other hand, a certain compensation for this. Vacuum tubes can be made to operate at exceedingly high speeds in applications other than computing machines, but these need not concern us here. In computing machines the maximum is a good deal lower, but it is still quite respectable. In the present state of the art, it is generally believed to be somewhere around a million actuations per second. The responses of a nerve cell are a good deal slower than this, perhaps 1/2000 of a second, and what really matters, the minimum time-interval required from stimulation to complete recovery and, possibly, renewed stimulation, is still longer than this—at best approximately 1/200 of a second. This gives a ratio of 1:5000, which, however, may be somewhat too favorable to the vacuum tube, since vacuum tubes, when used as switching organs at the 1,000,000 steps per second rate, are practically never run at a 100 per cent duty cycle. A ratio like 1:2000 would, therefore, seem to be more equitable. Thus the vacuum tube, at something like a billion times the expense, outperforms the neuron by a factor of somewhat over 1000. There is, therefore, some justice in saying that it is less efficient by a factor of the order of a million.

The basic fact is, in every respect, the small size of the neuron compared to the vacuum tube. This ratio is about a billion, as pointed out above. What is it due to?

Analysis of the Reasons for the Extreme Ratio of Sizes. The origin of this discrepancy lies in the fundamental control organ or, rather, control arrangement of the vacuum tube as compared to that of the neuron. In the vacuum tube the critical area of control is the space between the cathode (where the active agents, the electrons, originate) and the grid (which controls the electron flow). This space is about one millimeter deep. The corresponding entity in a neuron is the wall of the nerve cell, the "membrane." Its thickness is about a micron (1/1000 millimeter), or somewhat less. At this point, therefore, there is a ratio of approximately 1:1000 in linear dimensions. This, by the way, is the main difference. The electrical fields, which exist in the controlling space, are about the same for the vacuum tube and for the neuron. The potential differences by which these organs can be reliably steered are tens of volts in one case and tens of millivolts in the other. Their ratio is again about 1:1000, and hence their gradients (the field strengths) are about identical. Now a ratio of 1:1000 in linear dimensions corresponds to a ratio of 1:1,000,000,000 in volume. Thus the discrepancy factor of a billion in 3-dimensional size (volume) corresponds, as it should, to a discrepancy factor of 1000 in linear size, that is, to the difference between the millimeter interelectrode-space depth of the vacuum tube and the micron membrane thickness of the neuron.

It is worth noting, although it is by no means surprising, how this divergence between objects, both of which are microscopic and are situated in the interior of the elementary components, leads to impressive macroscopic differences between the organisms built upon them. This difference between a millimeter object and a micron object causes the ENIAC to weigh 30 tons and to dissipate 150 kilowatts of energy, while the human central nervous system, which is functionally about a million times larger, has the weight of the order of a pound and is accommodated within the human skull. In assessing the weight and size of the ENIAC as stated above, we should also remember that this huge apparatus is needed in order to handle 20 numbers of 10 decimals each, that is, a total of 200 decimal digits, the equivalent of about 700 binary digits—merely 700 simultaneous pieces of "yes-no" information!

Technological Interpretation of These Reasons. These considerations should make it clear that our present technology is still very imperfect in handling information at high speed and high degrees of complexity. The apparatus which results is simply enormous, both physically and in its energy requirements.

The weakness of this technology lies probably, in part at least, in the materials employed. Our present techniques involve the using of metals, with rather close spacings, and at certain critical points separated by vacuum only. This combination of media has a peculiar mechanical instability that is entirely alien to living nature. By this I mean the simple fact that, if a living organism is mechanically injured, it has a strong tendency to restore itself. If, on the other hand, we hit a man-made mechanism with a sledge hammer, no such restoring tendency is apparent. If two pieces of metal are close together, the small vibrations and other mechanical disturbances, which always exist in the ambient medium, constitute a risk in that they may bring them into contact. If they were at different electrical potentials, the next thing that may happen after this short circuit is that they can become electrically soldered together and the contact becomes permanent. At this point, then, a genuine and permanent breakdown will have occurred. When we injure the membrane of a nerve cell, no such thing happens. On the contrary, the membrane will usually reconstitute itself after a short delay.

It is this mechanical instability of our materials which prevents us from reducing sizes further. This instability and other phenomena of a comparable character make the behavior in our componentry less than wholly reliable, even at the present sizes. Thus it is the inferiority of our materials, compared with those used in nature, which prevents us from attaining the high degree of complication and the small dimensions which have been attained by natural organisms.

THE FUTURE LOGICAL THEORY OF AUTOMATA

Further Discussion of the Factors That Limit the Present Size of Artificial Automata. We have emphasized how the complication is limited in artificial automata, that is, the complication which can be handled without extreme difficulties and for which automata can still be expected to function reliably. Two reasons that put a limit on complication in this sense have already been given. They are the large size and the limited reliability of the componentry that we must use, both of them due to the fact that we are employing materials which seem to be quite satisfactory in simpler applications, but marginal and inferior to the natural ones in this highly complex application. There is, however, a third important limiting factor, and we should now turn our attention to it. This factor is of an intellectual, and not physical, character.

The Limitation Which Is Due to the Lack of a Logical Theory of Automata. We are very far from possessing a theory of automata which deserves that name, that is, a properly mathematical-logical theory.

There exists today a very elaborate system of formal logic, and, specifically, of logic as applied to mathematics. This is a discipline with many good sides, but also with certain serious weaknesses. This is not the occasion to enlarge upon the good sides, which I have certainly no intention to belittle. About the inadequacies, however, this may be said: Everybody who has worked in formal logic will confirm that it is one of the technically most refractory parts of mathematics. The reason for this is that it deals with rigid, all-or-none concepts, and has very little contact with the continuous concept of the real or of the complex number, that is, with mathematical analysis. Yet analysis is the technically most successful and best-elaborated part of mathematics. Thus formal logic is, by the nature of its approach, cut off from the best cultivated portions of mathematics, and forced onto the most difficult part of the mathematical terrain, into combinatorics.

The theory of automata, of the digital, all-or-none type, as discussed up to now, is certainly a chapter in formal logic. It would, therefore, seem that it will have to share this unattractive property of formal logic. It will have to be, from the mathematical point of view, combinatorial rather than analytical.

Probable Characteristics of Such a Theory. Now it seems to me that this will in fact not be the case. In studying the functioning of automata, it is clearly necessary to pay attention to a circumstance which has never before made its appearance in formal logic.

Throughout all modern logic, the only thing that is important is whether a result can be achieved in a finite number of elementary steps or not. The size of the number of steps which are required, on the other hand, is hardly ever a concern of formal logic. Any finite sequence of correct steps is, as a matter of principle, as good as any other. It is a matter of no consequence whether the number is small or large, or even so large that it couldn't possibly be carried out in a lifetime, or in the presumptive lifetime of the stellar universe as we know it. In dealing with automata, this statement must be significantly modified. In the case of an automaton the thing which matters is not only whether it can reach a certain result in a finite number of steps at all but also how many such steps are needed. There are two reasons. First, automata are constructed in order to reach certain results in certain pre-assigned durations, or at least in pre-assigned orders of magnitude of duration. Second, the componentry employed has on every individual operation a small but nevertheless non-zero probability of failing. In a sufficiently long chain of operations the cumulative effect of these individual probabilities of failure may (if unchecked) reach the order of magnitude of unity—at which point it produces, in effect, complete unreliability. The probability levels which are involved here are very low, but still not too far removed from the domain of ordinary technological experience. It is not difficult to estimate that a high-speed computing machine, dealing with a typical problem, may have to perform as much as 10^{12} individual operations. The probability of error on an individual operation which can be tolerated must, therefore, be small compared to 10^{-12}. I might mention that an electromechanical relay (a telephone relay) is at present considered acceptable if its probability of failure on an individual operation is of the order 10^{-8}. It is considered excellent if this order of probability is 10^{-9}. Thus the reliabilities required in a high-speed computing machine are higher, but not prohibitively higher, than those that constitute sound practice in certain existing industrial fields. The actually obtainable reliabilities are, however, not likely to leave a very wide margin against the minimum requirements just mentioned. An exhaustive study and a non-trivial theory will, therefore, certainly be called for.

Thus the logic of automata will differ from the present system of formal logic in two relevant respects.

1. The actual length of "chains of reasoning," that is, of the chains of operations, will have to be considered.

2. The operations of logic (syllogisms, conjunctions, disjunctions, negations, etc., that is, in the terminology that is customary for automata, various forms of gating, coincidence, anti-coincidence, blocking, etc., actions) will all have to be treated by procedures which allow exceptions (malfunctions) with low but non-zero probabilities. All of this will lead to theories which are much less rigidly of an all-or-none nature than past and present formal logic. They will be of a much less combinatorial, and much more analytical, character. In fact, there are numerous indications to make us believe that this new system of formal logic will move closer to another discipline which has been little linked in the past with logic. This is thermodynamics, primarily in the form it was received from Boltzmann, and is that part of theoretical physics which comes nearest in some of its aspects to manipulating and measuring information. Its techniques are indeed much more analytical than combinatorial, which again illustrates the point that I have been trying to make above. It would, however, take me too far to go into this subject more thoroughly on this occasion.

All of this re-emphasizes the conclusion that was indicated earlier, that a detailed, highly mathematical, and more specifically analytical, theory of automata and of information is needed. We possess only the first indications of such a theory at present. In assessing artificial automata, which are, as I discussed earlier, of only moderate size, it has been possible to get along in a rough, empirical manner without such a theory. There is every reason to believe that this will not be possible with more elaborate automata.

Effects of the Lack of a Logical Theory of Automata on the Procedures in Dealing with Errors. This, then, is the last, and very important, limiting factor. It is unlikely that we could construct automata of a much higher complexity than the ones we now have, without possessing a very advanced and subtle theory of automata and information. A fortiori, this is inconceivable for automata of such enormous complexity as is possessed by the human central nervous system.

This intellectual inadequacy certainly prevents us from getting much farther than we are now.

A simple manifestation of this factor is our present relation to error checking. In living organisms malfunctions of components occur. The organism obviously has a way to detect them and render them harmless. It is easy to estimate that the number of nerve actuations which occur in a normal lifetime must be of the order of 10^{20}. Obviously, during this chain of events there never occurs a malfunction which cannot be corrected by the organism itself, without any significant outside intervention. The system must, therefore, contain the necessary arrangements to diagnose errors as they occur, to readjust the organism so as to minimize the effects of the errors, and finally to correct or to block permanently the faulty components. Our modus procedendi with respect to malfunctions in our artificial automata is entirely different. Here the actual practice, which has the consensus of all experts of the field, is somewhat like this: Every effort is made to detect (by mathematical or by automatical checks) every error as soon as it occurs. Then an attempt is made to isolate the component that caused the error as rapidly as feasible. This may be done partly automatically, but in any case a significant part of this diagnosis must be effected by intervention from the outside. Once the faulty component has been identified, it is immediately corrected or replaced.

Note the difference in these two attitudes. The basic principle of dealing with malfunctions in nature is to make their effect as unimportant as possible and to apply correctives, if they are necessary at all, at leisure. In our dealings with artificial automata, on the other hand, we require an immediate diagnosis. Therefore, we are trying to arrange the automata in such a manner that errors will become as conspicuous as possible, and intervention and correction follow immediately. In other words, natural organisms are constructed to make errors as inconspicuous, as harmless, as possible. Artificial automata are designed to make errors as conspicuous, as disastrous, as possible. The rationale of this difference is not far to seek. Natural organisms are sufficiently well conceived to be able to operate even when malfunctions have set in. They can operate in spite of malfunctions, and their subsequent tendency is to remove these malfunctions. An artificial automaton could certainly be designed so as to be able to operate normally in spite of a limited number of malfunctions in certain limited areas. Any malfunction, however, represents a considerable risk that some generally degenerating process has already set in within the machine. It is, therefore, necessary to intervene immediately, because a machine which has begun to malfunction has only rarely a tendency to restore itself, and will more probably go from bad to worse. All of this comes back to one thing. With our artificial automata we are moving much more in the dark than nature appears to be with its organisms. We are, and apparently, at least at present, have to be, much more "scared" by the occurrence of an isolated error and by the malfunction which must be behind it. Our behavior is clearly that of overcaution, generated by ignorance.

The Single-Error Principle. A minor side light to this is that almost all our error-diagnosing techniques are based on the assumption that the machine contains only one faulty component. In this case, iterative subdivisions of the machine into parts permit us to determine which portion contains the fault. As soon as the possibility exists that the machine may contain several faults, these, rather powerful, dichotomic methods of diagnosis are lost. Error diagnosing then becomes an increasingly hopeless proposition. The high premium on keeping the number of errors to be diagnosed down to one, or at any rate as low as possible, again illustrates our ignorance in this field, and is one of the main reasons why errors must be made as conspicuous as possible, in order to be recognized and apprehended as soon after their occurrence as feasible, that is, before further errors have had time to develop.

PRINCIPLES OF DIGITALIZATION

Digitalization of Continuous Quantities: the Digital Expansion Method and the Counting Method. Consider the digital part of a natural organism; specifically, consider the nervous system. It seems that we are indeed justified in assuming that this is a digital mechanism, that it transmits messages which are made up of signals possessing the all-or-none character. (See also the earlier discussion, page 10.)

In other words, each elementary signal, each impulse, simply either is or is not there, with no further shadings. A particularly relevant illustration of this fact is furnished by those cases where the underlying problem has the opposite character, that is, where the nervous system is actually called upon to transmit a continuous quantity. Thus the case of a nerve which has to report on the value of a pressure is characteristic.

Assume, for example, that a pressure (clearly a continuous quantity) is to be transmitted. It is well known how this trick is done. The nerve which does it still transmits nothing but individual all-or-none impulses. How does it then express the continuously numerical value of pressure in terms of these impulses, that is, of digits? In other words, how does it encode a continuous number into a digital notation? It does certainly not do it by expanding the number in question into decimal (or binary, or any other base) digits in the conventional sense. What appears to happen is that it transmits pulses at a frequency which varies and which is within certain limits proportional to the continuous quantity in question, and generally a monotone function of it. The mechanism which achieves this "encoding" is, therefore, essentially a frequency modulation system.

The details are known. The nerve has a finite recovery time. In other words, after it has been pulsed once, the time that has to lapse before another stimulation is possible is finite and dependent upon the strength of the ensuing (attempted) stimulation. Thus, if the nerve is under the influence of a continuing stimulus (one which is uniformly present at all times, like the pressure that is being considered here), then the nerve will respond periodically, and the length of the period between two successive stimulations is the recovery time referred to earlier, that is, a function of the strength of the constant stimulus (the pressure in the present case). Thus, under a high pressure, the nerve may be able to respond every 8 milliseconds, that is, transmit at the rate of 125 impulses per second; while under the influence of a smaller pressure it may be able to repeat only every 14 milliseconds, that is, transmit at the rate of 71 times per second. This is very clearly the behavior of a genuinely yes-or-no organ, of a digital organ. It is very instructive, however, that it uses a "count" rather than a "decimal expansion" (or "binary expansion," etc.) method.

Comparison of the Two Methods. The Preference of Living Organisms for the Counting Method. Compare the merits and demerits of these two methods. The counting method is certainly less efficient than the expansion method. In order to express a number of about a million (that is, a physical quantity of a million distinguishable resolution-steps) by counting, a million pulses have to be transmitted. In order to express a number of the same size by expansion, 6 or 7 decimal digits are needed, that is, about 20 binary digits. Hence, in this case only 20 pulses are needed. Thus our expansion method is much more economical in notation than the counting methods which are resorted to by nature. On the other hand, the counting method has a high stability and safety from error. If you express a number of the order of a million by counting and miss a count, the result is only irrelevantly changed. If you express it by (decimal or binary) expansion, a single error in a single digit may vitiate the entire result. Thus the undesirable trait of our computing machines reappears in our digital expansion system; in fact, the former is clearly deeply connected with, and partly a consequence of, the latter. The high stability and nearly error-proof character of natural organisms, on the other hand, is reflected in the counting method that they seem to use in this case. All of this reflects a general rule. You can increase the safety from error by a reduction of the efficiency of the notation, or, to say it positively, by allowing redundancy of notation. Obviously, the simplest form of achieving safety by redundancy is to use the, per se, quite unsafe digital expansion notation, but to repeat every such message several times. In the case under discussion, nature has obviously resorted to an even more redundant and even safer system.

There are, of course, probably other reasons why the nervous system uses the counting rather than the digital expansion. The encoding-decoding facilities required by the former are much simpler than those required by the latter. It is true, however, that nature seems to be willing and able to go much further in the direction of complication than we are, or rather than we can afford to go. One may, therefore, suspect that if the only demerit of the digital expansion system were its greater logical complexity, nature would not, for this reason alone, have rejected it. It is, nevertheless, true that we have nowhere an indication of its use in natural organisms. It is difficult to tell how much "final" validity one should attach to this observation. The point deserves at any rate attention, and should receive it in future investigations of the functioning of the nervous system.

FORMAL NEURAL NETWORKS

The McCulloch-Pitts Theory of Formal Neural Networks. A great deal more could be said about these things from the logical and the organizational point of view, but I shall not attempt to say it here. I shall instead go on to discuss what is probably the most significant result obtained with the axiomatic method up to now. I mean the remarkable theorems of McCulloch and Pitts on the relationship of logics and neural networks.

In this discussion I shall, as I have said, take the strictly axiomatic point of view. I shall, therefore, view a neuron as a "black box" with a certain number of inputs that receive stimuli and an output that emits stimuli. To be specific, I shall assume that the input connections of each one of these can be of two types, excitatory and inhibitory. The boxes themselves are also of two types, threshold 1 and threshold 2. These concepts are linked and circumscribed by the following definitions. In order to stimulate such an organ it is necessary that it should receive simultaneously at least as many stimuli on its excitatory inputs as correspond to its threshold, and not a single stimulus on any one of its inhibitory inputs. If it has been thus stimulated, it will after a definite time delay (which is assumed to be always the same, and may be used to define the unit of time) emit an output pulse. This pulse can be taken by appropriate connections to any number of inputs of other neurons (also to any of its own inputs) and will produce at each of these the same type of input stimulus as the ones described above.

It is, of course, understood that this is an oversimplification of the actual functioning of a neuron. I have already discussed the character, the limitations, and the advantages of the axiomatic method. (See pages 2 and 10.) They all apply here, and the discussion which follows is to be taken in this sense.

McCulloch and Pitts have used these units to build up complicated networks which may be called "formal neural networks." Such a system is built up of any number of these units, with their inputs and outputs suitably interconnected with arbitrary complexity. The "functioning" of such a network may be defined by singling out some of the inputs of the entire system and some of its outputs, and then describing what original stimuli on the former are to cause what ultimate stimuli on the latter.

The Main Result of the McCulloch-Pitts Theory. McCulloch and Pitts' important result is that any functioning in this sense which can be defined at all logically, strictly, and unambiguously in a finite number of words can also be realized by such a formal neural network.

It is well to pause at this point and to consider what the implications are. It has often been claimed that the activities and functions of the human nervous system are so complicated that no ordinary mechanism could possibly perform them. It has also been attempted to name specific functions which by their nature exhibit this limitation. It has been attempted to show that such specific functions, logically, completely described, are per se unable of mechanical, neural realization. The McCulloch-Pitts result puts an end to this. It proves that anything that can be exhaustively and unambiguously described, anything that can be completely and unambiguously put into words, is ipso facto realizable by a suitable finite neural network. Since the converse statement is obvious, we can therefore say that there is no difference between the possibility of describing a real or imagined mode of behavior completely and unambiguously in words, and the possibility of realizing it by a finite formal neural network. The two concepts are co-extensive. A difficulty of principle embodying any mode of behavior in such a network can exist only if we are also unable to describe that behavior completely.

Thus the remaining problems are these two. First, if a certain mode of behavior can be effected by a finite neural network, the question still remains whether that network can be realized within a practical size, specifically, whether it will fit into the physical limitations of the organism in question. Second, the question arises whether every existing mode of behavior can really be put completely and unambiguously into words.

The first problem is, of course, the ultimate problem of nerve physiology, and I shall not attempt to go into it any further here. The second question is of a different character, and it has interesting logical connotations.

Interpretations of This Result. There is no doubt that any special phase of any conceivable form of behavior can be described "completely and unambiguously" in words. This description may be lengthy, but it is always possible. To deny it would amount to adhering to a form of logical mysticism which is surely far from most of us. It is, however, an important limitation, that this applies only to every element separately, and it is far from clear how it will apply to the entire syndrome of behavior. To be more specific, there is no difficulty in describing how an organism might be able to identify any two rectilinear triangles, which appear on the retina, as belonging to the same category "triangle." There is also no difficulty in adding to this, that numerous other objects, besides regularly drawn rectilinear triangles, will also be classified and identified as triangles—triangles whose sides are curved, triangles whose sides are not fully drawn, triangles that are indicated merely by a more or less homogeneous shading of their interior, etc. The more completely we attempt to describe everything that may conceivably fall under this heading, the longer the description becomes. We may have a vague and uncomfortable feeling that a complete catalogue along such lines would not only be exceedingly long, but also unavoidably indefinite at its boundaries. Nevertheless, this may be a possible operation.

All of this, however, constitutes only a small fragment of the more general concept of identification of analogous geometrical entities. This, in turn, is only a microscopic piece of the general concept of analogy. Nobody would attempt to describe and define within any practical amount of space the general concept of analogy which dominates our interpretation of vision. There is no basis for saying whether such an enterprise would require thousands or millions or altogether impractical numbers of volumes. Now it is perfectly possible that the simplest and only practical way actually to say what constitutes a visual analogy consists in giving a description of the connections of the visual brain. We are dealing here with parts of logics with which we have practically no past experience. The order of complexity is out of all proportion to anything we have ever known. We have no right to assume that the logical notations and procedures used in the past are suited to this part of the subject. It is not at all certain that in this domain a real object might not constitute the simplest description of itself, that is, any attempt to describe it by the usual literary or formal-logical method may lead to something less manageable and more involved. In fact, some results in modern logic would tend to indicate that phenomena like this have to be expected when we come to really complicated entities. It is, therefore, not at all unlikely that it is futile to look for a precise logical concept, that is, for a precise verbal description, of "visual analogy." It is possible that the connection pattern of the visual brain itself is the simplest logical expression or definition of this principle.

Obviously, there is on this level no more profit in the McCulloch-Pitts result. At this point it only furnishes another illustration of the situation outlined earlier. There is an equivalence between logical principles and their embodiment in a neural network, and while in the simpler cases the principles might furnish a simplified expression of the network, it is quite possible that in cases of extreme complexity the reverse is true.

All of this does not alter my belief that a new, essentially logical, theory is called for in order to understand high-complication automata and, in particular, the central nervous system. It may be, however, that in this process logic will have to undergo a pseudomorphosis to neurology to a much greater extent than the reverse. The foregoing analysis shows that one of the relevant things we can do at this moment with respect to the theory of the central nervous system is to point out the directions in which the real problem does not lie.

THE CONCEPT OF COMPLICATION; SELF-REPRODUCTION

The Concept of Complication. The discussions so far have shown that high complexity plays an important role in any theoretical effort relating to automata, and that this concept, in spite of its prima facie quantitative character, may in fact stand for something qualitative—for a matter of principle. For the remainder of my discussion I will consider a remoter implication of this concept, one which makes one of the qualitative aspects of its nature even more explicit.

There is a very obvious trait, of the "vicious circle" type, in nature, the simplest expression of which is the fact that very complicated organisms can reproduce themselves.

We are all inclined to suspect in a vague way the existence of a concept of "complication." This concept and its putative properties have never been clearly formulated. We are, however, always tempted to assume that they will work in this way. When an automaton performs certain operations, they must be expected to be of a lower degree of complication than the automaton itself. In particular, if an automaton has the ability to construct another one, there must be a decrease in complication as we go from the parent to the construct. That is, if A can produce B, then A in some way must have contained a complete description of B. In order to make it effective, there must be, furthermore, various arrangements in A that see to it that this description is interpreted and that the constructive operations that it calls for are carried out. In this sense, it would therefore seem that a certain degenerating tendency must be expected, some decrease in complexity as one automaton makes another automaton.

Although this has some indefinite plausibility to it, it is in clear contradiction with the most obvious things that go on in nature. Organisms reproduce themselves, that is, they produce new organisms with no decrease in complexity. In addition, there are long periods of evolution during which the complexity is even increasing. Organisms are indirectly derived from others which had lower complexity.

Thus there exists an apparent conflict of plausibility and evidence, if nothing worse. In view of this, it seems worth while to try to see whether there is anything involved here which can be formulated rigorously.

So far I have been rather vague and confusing, and not unintentionally at that. It seems to me that it is otherwise impossible to give a fair impression of the situation that exists here. Let me now try to become specific.

Turing's Theory of Computing Automata. The English logician, Turing, about twelve years ago attacked the following problem.

He wanted to give a general definition of what is meant by a computing automaton. The formal definition came out as follows:

An automaton is a "black box," which will not be described in detail but is expected to have the following attributes. It possesses a finite number of states, which need be prima facie characterized only by stating their number, say n, and by enumerating them accordingly: $1, 2, \cdots n$. The essential operating characteristic of the automaton consists of describing how it is caused to change its state, that is, to go over from a state i into a state j. This change requires some interaction with the outside world, which will be standardized in the following manner. As far as the machine is concerned, let the whole outside world consist of a long paper tape. Let this tape be, say, 1 inch wide, and let it be subdivided into fields (squares) 1 inch long. On each field of this strip we may or may not put a sign, say, a dot, and it is assumed that it is possible to erase as well as to write in such a dot. A field marked with a dot will be called a "1," a field unmarked with a dot will be called a "0." (We might permit more ways of marking, but Turing showed that this is irrelevant and does not lead to any essential gain in generality.) In describing the position of the tape relative to the automaton it is assumed that one particular field of the tape is under direct inspection by the automaton, and that the automaton has the ability to move the tape forward and backward, say, by one field at a time. In specifying this, let the automaton be in the state i $(= 1 \cdots, n)$, and let it see on the tape an e $(= 0, 1)$. It will then go over into the state j $(= 0, 1, \cdots, n)$, move the tape by p fields ($p = 0, +1, -1$; $+1$ is a move forward, -1 is a move backward), and inscribe into the new field that it sees f ($= 0, 1$; inscribing 0 means erasing; inscribing 1 means putting in a dot). Specifying j, p, f as functions of i, e is then the complete definition of the functioning of such an automaton.

Turing carried out a careful analysis of what mathematical processes can be effected by automata of this type. In this connection he proved various theorems concerning the classical "decision problem" of logic, but I shall not go into these matters here. He did, however, also introduce and analyze the concept of a "universal automaton," and this is part of the subject that is relevant in the present context.

An infinite sequence of digits e $(= 0, 1)$ is one of the basic entities in mathematics. Viewed as a binary expansion, it is essentially equivalent to the concept of a real number. Turing, therefore, based his consideration on these sequences.

He investigated the question as to which automata were able to construct which sequences. That is, given a definite law for the formation of such a sequence, he inquired as to which automata can be used to form the sequence based on that law. The process of "forming" a sequence is interpreted in this manner. An automaton is able to "form" a certain sequence if it is possible to specify a finite length of tape, appropriately marked, so that, if this tape is fed to the automaton in question, the automaton will thereupon write the sequence on the remaining (infinite) free portion of the tape. This process of writing the infinite sequence is, of course, an indefinitely continuing one. What is meant is that the automaton will keep running indefinitely and, given a sufficiently long time, will have inscribed any desired (but of course finite) part of the (infinite) sequence. The finite, premarked, piece of tape constitutes the "instruction" of the automaton for this problem.

An automaton is "universal" if any sequence that can be produced by any automaton at all can also be solved by this particular automaton. It will, of course, require in general a different instruction for this purpose.

The Main Result of the Turing Theory. We might expect a priori that this is impossible. How can there be an automaton which is at least as effective as any conceivable automaton, including, for example, one of twice its size and complexity?

Turing, nevertheless, proved that this is possible. While his construction is rather involved, the underlying principle is nevertheless quite simple. Turing observed that a completely general description of any conceivable automaton can be (in the sense of the foregoing definition) given in a finite number of words. This description will contain certain empty passages—those referring to the functions mentioned earlier (j, p, f in terms of i, e), which specify the actual functioning of the automaton. When these empty passages are filled in, we deal with a specific automaton. As long as they are left empty, this schema represents the general definition of the general automaton. Now it becomes possible to describe an automaton which has the ability to interpret such a definition. In other words, which, when fed the functions that in the sense described above define a specific automaton, will thereupon function like the object described. The ability to do this is no more mysterious than the ability to read a dictionary and a grammar and to follow their instructions about the uses and principles of combinations of words. This automaton, which is constructed to read a description and to imitate the object described, is then the universal automaton in the sense of Turing. To make it duplicate any operation that any other automaton can perform, it suffices to furnish it with a description of the automaton in question and, in addition, with the instructions which that device would have required for the operation under consideration.

Broadening of the Program to Deal with Automata That Produce Automata. For the question which concerns me here, that of "self-reproduction" of automata, Turing's procedure is too narrow in one respect only. His automata are purely computing machines. Their output is a piece of tape with zeros and ones on it. What is needed for the construction to which I referred is an automaton whose output is other automata. There is, however, no difficulty in principle in dealing with this broader concept and in deriving from it the equivalent of Turing's result.

The Basic Definitions. As in the previous instance, it is again of primary importance to give a rigorous definition of what constitutes an automaton for the purpose of the investigation. First of all, we have to draw up a complete list of the elementary parts to be used. This list must contain not only a complete enumeration but also a complete operational definition of each elementary part. It is relatively easy to draw up such a list, that is, to write a catalogue of "machine parts" which is sufficiently inclusive to permit the construction of the wide variety of mechanisms here required, and which has the axiomatic rigor that is needed for this kind of consideration. The list need not be very long either. It can, of course, be made either arbitrarily long or arbitrarily short. It may be lengthened by including in it, as elementary parts, things which could be achieved by combinations of others. It can be made short—in fact, it can be made to consist of a single unit—by endowing each elementary part with a multiplicity of attributes and functions. Any statement on the number of elementary parts required will therefore represent a common-sense compromise, in which nothing too complicated is expected from any one elementary part, and no elementary part is made to perform several, obviously separate, functions. In this sense, it can be shown that about a dozen elementary parts suffice. The problem of self-reproduction can then be stated like this: Can one build an aggregate out of such elements in such a manner that if it is put into a reservoir, in which there float all these elements in large numbers, it will then begin to construct other aggregates, each of which will at the end turn out to be another automaton exactly like the original one? This is feasible, and the principle on which it can be based is closely related to Turing's principle outlined earlier.

Outline of the Derivation of the Theorem Regarding Self-reproduction. First of all, it is possible to give a complete description of everything that is an automaton in the sense considered here. This description is to be conceived as a general one, that is, it will again contain empty spaces. These empty spaces have to be filled in with the functions which describe the actual structure of an automaton. As before, the difference between these spaces filled and unfilled is the difference between the description of a specific automaton and the general description of a general automaton. There is no difficulty of principle in describing the following automata.

(*a*) Automaton *A*, which when furnished the description of any other automaton in terms of appropriate functions, will construct that entity. The description should in this case not be given in the form of a marked tape, as in Turing's case, because we will not normally choose a tape as a structural element. It is quite easy, however, to describe combinations of structural elements which have all the notational properties of a tape with fields that can be marked. A description in this sense will be called an instruction and denoted by a letter *I*.

"Constructing" is to be understood in the same sense as before. The constructing automaton is supposed to be placed in a reservoir in which all elementary components in large numbers are floating, and it will effect its construction in that milieu. One need not worry about how a fixed automaton of this sort can produce others which are larger and more complex than itself. In this case the greater size and the higher complexity of the object to be constructed will be reflected in a presumably still greater size of the instructions *I* that have to be furnished. These instructions, as pointed out, will have to be aggregates of elementary parts. In this sense, certainly, an entity will enter the process whose size and complexity is determined by the size and complexity of the object to be constructed.

In what follows, all automata for whose construction the facility *A* will be used are going to share with *A* this property. All of them will have a place for an instruction *I*, that is, a place where such an instruction can be inserted. When such an automaton is being described (as, for example, by an appropriate instruction), the specification of the location for the insertion of an instruction *I* in the foregoing sense is understood to form a part of the description. We may, therefore, talk of "inserting a given instruction *I* into a given automaton," without any further explanation.

(*b*) Automaton *B*, which can make a copy of any instruction *I* that is furnished to it. *I* is an aggregate of elementary parts in the sense outlined in (*a*), replacing a tape. This facility will be used when I furnishes a description of another automaton. In other words, this automaton is nothing more subtle than a "reproducer"—the machine which can read a punched tape and produce a second punched tape that is identical with the first. Note that this automaton, too, can produce objects which are larger and more complicated than itself. Note again that there is nothing surprising about it. Since it can only copy, an object of the exact size and complexity of the output will have to be furnished to it as input.

After these preliminaries, we can proceed to the decisive step.

(*c*) Combine the automata A and B with each other, and with a control mechanism C which does the following. Let A be furnished with an instruction I (again in the sense of [*a*] and [*b*]). Then C will first cause A to construct the automaton which is described by this instruction I. Next C will cause B to copy the instruction I referred to above, and insert the copy into the automaton referred to above, which has just been constructed by A. Finally, C will separate this construction from the system $A + B + C$ and "turn it loose" as an independent entity.

(*d*) Denote the total aggregate $A + B + C$ by D.

(*e*) In order to function, the aggregate $D = A + B + C$ must be furnished with an instruction I, as described above. This instruction, as pointed out above, has to be inserted into A. Now form an instruction I_D, which describes this automaton D, and insert I_D into A within D. Call the aggregate which now results E.

E is clearly self-reproductive. Note that no vicious circle is involved. The decisive step occurs in E, when the instruction I_D, describing D, is constructed and attached to D. When the construction (the copying) of I_D is called for, D exists already, and it is in no wise modified by the construction of I_D. I_D is simply added to form E. Thus there is a definite chronological and logical order in which D and I_D have to be formed, and the process is legitimate and proper according to the rules of logic.

Interpretations of This Result and of Its Immediate Extensions. The description of this automaton E has some further attractive sides, into which I shall not go at this time at any length. For instance, it is quite clear that the instruction I_D is roughly effecting the functions of a gene. It is also clear that the copying mechanism B performs the fundamental act of reproduction, the duplication of the genetic material, which is clearly the fundamental operation in the multiplication of living cells. It is also easy to see how arbitrary alterations of the system E, and in particular of I_D, can exhibit certain typical traits which appear in connection with mutation, lethally as a rule, but with a possibility of continuing reproduction with a modification of traits. It is, of course, equally clear at which point the analogy ceases to be valid. The natural gene does probably not contain a complete description of the object whose construction its presence stimulates. It probably contains only general pointers, general cues. In the generality in which the foregoing consideration is moving, this simplification is not attempted. It is, nevertheless, clear that this simplification, and others similar to it, are in themselves of great and qualitative importance. We are very far from any real understanding of the natural processes if we do not attempt to penetrate such simplifying principles.

Small variations of the foregoing scheme also permit us to construct automata which can reproduce themselves and, in addition, construct others. (Such an automaton performs more specifically what is probably a—if not the—typical gene function, self-reproduction plus production—or stimulation of production—of certain specific enzymes.) Indeed, it suffices to replace the I_D by an instruction I_{D+F}, which describes the automaton D plus another given automaton F. Let D, with I_{D+F} inserted into A within it, be designated by E_F. This E_F clearly has the property already described. It will reproduce itself, and, besides, construct F.

Note that a "mutation" of E_F, which takes place within the F-part of I_{D+F} in E_F, is not lethal. If it replaces F by F', it changes E_F into $E_{F'}$, that is, the "mutant" is still self-reproductive; but its by-product is changed—F' instead of F. This is, of course, the typical non-lethal mutant.

All these are very crude steps in the direction of a systematic theory of automata. They represent, in addition, only one particular direction. This is, as I indicated before, the direction towards forming a rigorous concept of what constitutes "complication." They illustrate that "complication" on its lower levels is probably degenerative, that is, that every automaton that can produce other automata will only be able to produce less complicated ones. There is, however, a certain minimum level where this degenerative characteristic ceases to be universal. At this point automata which can reproduce themselves, or even construct higher entities, become possible. This fact, that complication, as well as organization, below a certain minimum level is degenerative, and beyond that level can become self-supporting and even increasing, will clearly play an important role in any future theory of the subject.

DISCUSSION

DR. MC CULLOCH: I confess that there is nothing I envy Dr. von Neumann more than the fact that the machines with which he has to cope are those for which he has, from the beginning, a blueprint of what the machine is supposed to do and how it is supposed to do it. Unfortunately for us in the biological sciences—or, at least, in psychiatry—we are presented with an alien, or enemy's, machine. We do not know exactly what the machine is supposed to do and certainly we have no blueprint of it. In attacking our problems, we only know, in psychiatry, that the machine is producing wrong answers. We know that, because of the damage by the machine to the machine itself and by its running amuck in the world. However, what sort of difficulty exists in that machine is no easy matter to determine.

As I see it what we need first and foremost is not a correct theory, but some theory to start from, whereby we may hope to ask a question so that we'll get an answer, if only to the effect that our notion was entirely erroneous. Most of the time we never even get around to asking the question in such a form that it can have an answer.

I'd like to say, historically, how I came to be interested in this particular problem, if you'll forgive me, because it does bear on this matter. I came, from a major interest in philosophy and mathematics, into psychology with the problem of how a thing like mathematics could ever arise—what sort of a thing it was. For that reason, I gradually shifted into psychology and thence, for the reason that I again and again failed to find the significant variables, I was forced into neurophysiology. The attempt to construct a theory in a field like this, so that it can be put to any verification, is tough. Humorously enough, I started entirely at the wrong angle, about 1919, trying to construct a logic for transitive verbs. That turned out to be as mean a problem as modal logic, and it was not until I saw Turing's paper that I began to get going the right way around, and with Pitts' help formulated the required logical calculus. What we thought we were doing (and I think we succeeded fairly well) was treating the brain as a Turing machine; that is, as a device which could perform the kind of functions which a brain must perform if it is only to go wrong and have a psychosis. The important thing was, for us, that we had to take a logic and subscript it for the time of the occurrence of a signal (which is, if you will, no more than a proposition on the move). This was needed in order to construct theory enough to be able to state how a nervous system could do anything. The delightful thing is that the very simplest set of appropriate assumptions is sufficient to show that a nervous system can compute any computable number. It is that kind of a device, if you like—a Turing machine.

The question at once arose as to how it did certain of the things that it did do. None of the theories tell you how a particular operation is carried out, any more than they tell you in what kind of a nervous system it is carried out, or any more than they tell you in what part of a computing machine it is carried out. For that you have to have the wiring diagram or the prescription for the relations of the gears.

This means that you are compelled to study anatomy, and to require of the anatomist the things he has rarely given us in sufficient detail. I taught neuro-anatomy while I was in medical school, but until the last year or two I have not been in a position to ask any neuro-anatomist for the precise detail of any structure. I had no physiological excuse for wanting that kind of information. Now we are beginning to need it.

DR. GERARD: I have had the privilege of hearing Dr. von Neumann speak on various occasions, and I always find myself in the delightful but difficult role of hanging on to the tail of a kite. While I can follow him, I can't do much creative thinking as we go along. I would like to ask one question, though, and suspect that it may be in the minds of others. You have carefully stated, at several points in your discourse, that anything that could be put into verbal form—into a question with words—could be solved. Is there any catch in this? What is the implication of just that limitation on the question?

DR. VON NEUMANN: I will try to answer, but my answer will have to be rather incomplete.

The first task that arises in dealing with any problem—more specifically, with any function of the central nervous system—is to formulate it unambiguously, to put it into words, in a rigorous sense. If a very complicated system—like the central nervous system—is involved, there arises the additional task of doing this "formulating," this "putting into words," with a number of words within reasonable limits—for example, that can be read in a lifetime. This is the place where the real difficulty lies.

In other words, I think that it is quite likely that one may give a purely descriptive account of the outwardly visible functions of the central nervous system in a humanly possible time. This may be 10 or 20 years—which is long, but not prohibitively long. Then, on the basis of the results of McCulloch and Pitts, one could draw within plausible time limitations a fictitious "nervous network" that can carry out all these functions. I suspect, however, that it will turn out to be much larger than the one that we actually possess. It is possible that it will prove to be too large to fit into the physical universe. What then? Haven't we lost the true problem in the process?

Thus the problem might better be viewed, not as one of imitating the functions of the central nervous system with just any kind of network, but rather as one of doing this with a network that will fit into the actual volume of the human brain. Or, better still, with one that can be kept going with our actual metabolistic "power supply" facilities, and that can be set up and organized by our actual genetic control facilities.

To sum up, I think that the first phase of our problem—the purely formalistic one, that one of finding any "equivalent network" at all—has been overcome by McCulloch and Pitts. I also think that much of the "malaise" felt in connection with attempts to "explain" the central nervous system belongs to this phase—and should therefore be considered removed. There remains, however, plenty of malaise due to the next phase of the problem, that one of finding an "equivalent network" of possible, or even plausible, dimensions and (metabolistic and genetic) requirements.

The problem, then, is not this: How does the central nervous system effect any one, particular thing? It is rather: How does it do all the things that it can do, in their full complexity? What are the principles of its organization? How does it avoid really serious, that is, lethal, malfunctions over periods that seem to average many decades?

DR. GERARD: Did you mean to imply that there are unformulated problems?

DR. VON NEUMANN: There may be problems which cannot be formulated with our present logical techniques.

DR. WEISS: I take it that we are discussing only a conceivable and logically consistent, but not necessarily real, mechanism of the nervous system. Any theory of the real nervous system, however, must explain the facts of regulation—that the mechanism will turn out the same or an essentially similar product even after the network of pathways has been altered in many unpredictable ways. According to von Neumann, a machine can be constructed so as to contain safeguards against errors and provision for correcting errors when they occur. In this case the future contingencies have been taken into account in constructing the machine. In the case of the nervous system, evolution would have had to build in the necessary corrective devices. Since the number of actual interferences and deviations produced by natural variation and by experimenting neurophysiologists is very great, I question whether a mechanism in which all these innumerable contingencies have been foreseen, and the corresponding corrective measures built in, is actually conceivable.

DR. VON NEUMANN: I will not try, of course, to answer the question as to how evolution came to any given point. I am going to make, however, a few remarks about the much more limited question regarding errors, foreseeing errors, and recognizing and correcting errors.

An artificial machine may well be provided with organs which recognize and correct errors automatically. In fact, almost every well-planned machine contains some organs whose function is to do just this—always within certain limited areas. Furthermore, if any particular machine is given, it is always possible to construct a second machine which "watches" the first one, and which senses and possibly even corrects its errors. The trouble is, however, that now the second machine's errors are unchecked, that is, *quis custodiet ipsos custodes?* Building a third, a fourth, etc., machine for second order, third order, etc., checking merely shifts the problem. In addition, the primary and the secondary machine will, together, make more errors than the first one alone, since they have more components.

Some such procedure on a more modest scale may nevertheless make sense. One might know, from statistical experience with a certain machine or class of machines, which ones of its components malfunction most frequently, and one may then "supervise" these only, etc.

Another possible approach, which permits a more general quantitative evaluation, is this: Assume that one had a machine which has a probability of 10^{-10} to malfunction on any single operation, that is, which will, on the average, make one error for any 10^{10} operations. Assume that this machine has to solve a problem that requires 10^{12} operations. Its normal "unsupervised" functioning will, therefore, on the average, give 100 errors in a single problem, that is, it will be completely unusable.

Connect now three such machines in such a manner that they always compare their results after every single operation, and then proceed as follows. (*a*) If all three have the same result, they continue unchecked. (*b*) If any two agree with each other, but not with the third, then all three continue with the value agreed on by the majority. (*c*) If no two agree with each other, then all three stop.

This system will produce a correct result, unless at some point in the problem two of the three machines err simultaneously. The probability of two given machines erring simultaneously on a given operation is $10^{-10} \times 10^{-10} = 10^{-20}$. The probability of any two doing this on a given operation is 3×10^{-20} (there are three possible pairs to be formed among three individuals [machines]). The probability of this happening at all (that is, anywhere) in the entire problem is $10^{12} \times 3 \times 10^{-20} = 3 \times 10^{-8}$, about one in 33 million.

Thus there is only one chance in 33 million that this triad of machines will fail to solve the problem correctly—although each member of the triad alone had hardly any chance to solve it correctly.

Note that this triad, as well as any other conceivable automatic contraption, no matter how sophisticatedly supervised, still offers a logical possibility of resulting error—although, of course, only with a low probability. But the incidence (that is, the probability) of error has been significantly lowered, and this is all that is intended.

DR. WEISS: In order to crystallize the issue, I want to reiterate that if you know the common types of errors that will occur in a particular machine, you can make provisions for the correction of these errors in constructing the machine. One of the major features of the nervous system, however, is its apparent ability to remedy situations that could not possibly have been foreseen. (The number of artificial interferences with the various apparatuses of the nervous system that can be applied without impairing the biologically useful response of the organism is infinite.) The concept of a nervous automaton should, therefore, not only be able to account for the normal operation of the nervous system but also for its relative stability under all kinds of abnormal situations.

DR. VON NEUMANN: I do not agree with this conclusion. The argumentation that you have used is risky, and requires great care.

One can in fact guard against errors that are not specifically foreseen. These are some examples that show what I mean.

One can design and build an electrical automaton which will function as long as every resistor in it deviates no more than 10 per cent from its standard design value. You may now try to disturb this machine by experimental treatments which will alter its resistor values (as, for example, by heating certain regions in the machine). As long as no resistor shifts by more than 10 per cent, the machine will function right—no matter how involved, how sophisticated, how "unforeseen" the disturbing experiment is.

Or—another example—one may develop an armor plate which will resist impacts up to a certain strength. If you now test it, it will stand up successfully in this test, as long as its strength limit is not exceeded, no matter how novel the design of the gun, propellant, and projectile used in testing, etc.

It is clear how these examples can be transposed to neural and genetic situations.

To sum up: Errors and sources of errors need only be foreseen generically, that is, by some decisive traits, and not specifically, that is, in complete detail. And these generic coverages may cover vast territories, full of unforeseen and unsuspected—but, in fine, irrelevant—details.

DR. MC CULLOCH: How about designing computing machines so that if they were damaged in air raids, or what not, they could replace parts, or service themselves, and continue to work?

DR. VON NEUMANN: These are really quantitative rather than qualitative questions. There is no doubt that one can design machines which, under suitable conditions, will repair themselves. A practical discussion is, however, rendered difficult by what I believe to be a rather accidental circumstance. This is, that we seem to be operating with much more unstable materials than nature does. A metal may seem to be more stable than a tissue, but, if a tissue is injured, it has a tendency to restore itself, while our industrial materials do not have this tendency, or have it to a considerably lesser degree. I don't think, however, that any question of principle is involved at this point. This reflects merely the present, imperfect state of our technology—a state that will presumably improve with time.

DR. LASHLEY: I'm not sure that I have followed exactly the meaning of "error" in this discussion, but it seems to me the question of precision of the organic machine has been somewhat exaggerated. In the computing machines, the one thing we demand is precision; on the other hand, when we study the organism, one thing which we never find is accuracy or precision. In any organic reaction there is a normal, or nearly normal, distribution of errors around a mean. The mechanisms of reaction are statistical in character and their accuracy is only that of a probability distribution in the activity of enormous numbers of elements. In this respect the organism resembles the analogical rather than the digital machine. The invention of symbols and the use of memorized number series convert the organism into a digital machine, but the increase in accuracy is acquired at the sacrifice of speed. One can estimate the number of books on a shelf at a glance, with some error. To count them requires much greater time. As a digital machine the organism is inefficient. That is why you build computing machines.

DR. VON NEUMANN: I would like to discuss this question of precision in some detail.

It is perfectly true that in all mathematical problems the answer is required with absolute rigor, with absolute reliability. This may, but need not, mean that it is also required with absolute precision.

In most problems for the sake of which computing machines are being built—mostly problems in various parts of applied mathematics, mathematical physics—the precision that is wanted is quite limited. That is, the data of the problem are only given to limited precision, and the result is only wanted to limited precision. This is quite compatible with absolute mathematical rigor, if the sensitivity of the result to changes in the data as well as the limits of uncertainty (that is, the amount of precision) of the result for given data are (rigorously) known.

The (input) data in physical problems are often not known to better than a few (say 5) per cent. The result may be satisfactory to even less precision (say 10 per cent). In this respect, therefore, the difference of outward precision requirements for an (artificial) computing machine and a (natural) organism need not at all be decisive. It is merely quantitative, and the quantitative factors involved need not be large at that.

The need for high precisions in the internal functioning of (artificial) computing machines is due to entirely different causes—and these may well be operating in (natural) organisms too. By this I do not mean that the arguments that follow should be carried over too literally to organisms. In fact, the "digital method" used in computing may be entirely alien to the nervous system. The discrete pulses used in neural communications look indeed more like "counting" by numeration than like a "digitalization." (In many cases, of course, they may express a logical code—this is quite similar to what goes on in computing machines.) I will, nevertheless, discuss the specifically "digital" procedure of our computing machine, in order to illustrate how subtle the distinction between "external" and "internal" precision requirements can be.

In a computing machine numbers may have to be dealt with as aggregates of 10 or more decimal places. Thus an internal precision of one in 10 billion or more may be needed, although the data are only good to one part in 20 (5 per cent), and the result is only wanted to one part in 10 (10 per cent). The reason for this strange discrepancy is that a fast machine will only be used on long and complicated problems. Problems involving 100 million multiplications will not be rarities. In a 4-decimal-place machine every multiplication introduces a "round-off" error of one part in 10,000; in a 6-place machine this is one part in a million; in a 10-place machine it is one part in 10 billion. In a problem of the size indicated above, such errors will occur 100 million times. They will be randomly distributed, and it follows therefore from the rules of mathematical statistics that the total error will probably not be 100 million times the individual (round-off) error, but about the square root of 100 million times, that is, about 10,000 times. A precision of 10 per cent—one part in 10—in the result should therefore require 10,000 times more precision than this on individual steps (multiplication round-offs): namely, one part in 100,000, that is, 5 decimal places. Actually, more will be required because the (round-off) errors made in the earlier parts of the calculation are frequently "amplified" by the operations of the subsequent parts of the calculation. For these reasons 8 to 10 decimal places are probably a minimum for such a machine, and actually many large problems may well require more.

Most analogy computing machines have much less precision than this (on elementary operations). The electrical ones usually one part in 100 or 1000, the best mechanical ones (the most advanced "differential analyzers") one part in 10,000 or 50,000. The virtue of the digital method is that it will, with componentry of very limited precision, give almost any precision on elementary operations. If one part in a million is wanted, one will use 6 decimal digits; if one part in 10 billions is wanted, one need only increase the number of decimal digits to 10; etc. And yet the individual components need only be able to distinguish reliably 10 different states (the 10 decimal digits from 0 to 9), and by some simple logical and organizational tricks one can even get along with components that can only distinguish two states!

I suspect that the central nervous system, because of the great complexity of its tasks, also faces problems of "internal" precision or reliability. The all-or-none character of nervous impulses may be connected with some technique that meets this difficulty, and this—unknown—technique might well be related to the digital system that we use in computing, although it is probably very different from the digital system in its technical details. We seem to have no idea as to what this technique is. This is again an indication of how little we know. I think, however, that the digital system of computing is the only thing known to us that holds any hope of an even remote affinity with that unknown, and merely postulated, technique.

DR. MC CULLOCH: I want to make a remark in partial answer to Dr. Lashley. I think that the major woe that I have always encountered in considering the behavior of organisms was not in such procedures as hitting a bull's-eye or judging a distance, but in mathematics and logic. After all, Vega did compute log tables to thirteen places. He made some four hundred and thirty errors, but the total precision of the work of that organism is simply incredible to me.

DR. LASHLEY: You must keep in mind that such an achievement is not the product of a single elaborate integration but represents a great number of separate processes which are, individually, simple discriminations far above threshold values and which do not require great accuracy of neural activity.

DR. HALSTEAD: As I listened to Dr. von Neumann's beautiful analysis of digital and analogous devices, I was impressed by the conceptual parsimony with which such systems may be described. We in the field of organic behavior are not yet so fortunate. Our parsimonies, for the most part, are still to be attained. There is virtually no class of behaviors which can at present be described with comparable precision. Whether such domains as thinking, intelligence, learning, emoting, language, perception, and response represent distinctive processes or only different attitudinal sets of the organism is by no means clear. It is perhaps for this reason that Dr. von Neumann did not specify the class or classes of behaviors which his automata simulate.

As Craik pointed out several years ago,[*] it isn't quite logically air-tight to compare the operations of models with highly specified ends with organic behaviors only loosely specified either hierarchically or as to ends. Craik's criterion was that our models must bear a proper "relation structure" to the steps in the processes simulated. The rules of the game are violated when we introduce gremlins, either good or bad gremlins, as intervening variables. It is not clear to me whether von Neumann means "noise" as a good or as a bad gremlin. I presume it is a bad one when it is desired to maximize "rationality" in the outcome. It is probable that rationality characterizes a restricted class of human behavior. I shall later present experimental evidence that the same normal or brain-injured man also produces a less restricted class of behavior which is "arational" if not irrational. I suspect that von Neumann biases his automata towards rationality by careful regulation of the energics of the substrate. Perhaps he would gain in similitude, however, were he to build unstable power supplies into his computers and observe the results.

It seems to me that von Neumann is approximating in his computers some of the necessary operations in the organic process recognized by psychologists under the term "abstraction." Analysis of this process of ordering to a criterion in brain-injured individuals suggests that three classes of outcome occur. First, there is the pure category (or "universal"); second, there is the partial category; and third, there is the phenomenal or non-recurrent organization. Operationalism restricts our concern to the first two classes. However, these define the third. It is probably significant that psychologists such as Spearman and Thurstone have made considerable progress in describing these outcomes in mathematical notation.

---

[*] *Nature of Explanation*, London, Cambridge University Press, 1943.

DR. LORENTE DE NÓ: I began my training in a very different manner from Dr. McCulloch. I began as an anatomist and became interested in physiology much later. Therefore, I am still very much of an anatomist, and visualize everything in anatomical terms. According to your discussion, Dr. von Neumann, of the McCulloch and Pitts automaton, anything that can be expressed in words can be performed by the automaton. To this I would say that I can remember what you said, but that the McCulloch-Pitts automaton could not remember what you said. No, the automaton does not function in the way that our nervous system does, because the only way in which that could happen, as far as I can visualize, is by having some change continuously maintained. Possibly the automaton can be made to maintain memory, but the automaton that does would not have the properties of our nervous system. We agree on that, I believe. The only thing that I wanted was to make the fact clear.

DR. VON NEUMANN: One of the peculiar features of the situation, of course, is that you can make a memory out of switching organs, but there are strong indications that this is not done in nature. And, by the way, it is not very efficient, as closer analysis shows.

Why the Mind Is in the Head

WARREN S. McCULLOCH
University of Illinois, College of Medicine, and
Illinois Neuropsychiatric Institute

As the industrial revolution concludes in bigger and better bombs, an intellectual revolution opens with bigger and better robots. The former revolution replaced muscles by engines and was limited by the law of the conservation of energy, or of mass-energy. The new revolution threatens us, the thinkers, with technological unemployment, for it will replace brains with machines limited by the law that entropy never decreases. These machines, whose evolution competition will compel us to foster, raise the appropriate practical question: "Why is the mind in the head?"

Coming as I do between psyche anatomized and psyche synthesized, I must so define my terms that I can bridge the traditional gulf between mind and body and the technical gap between things begotten and things made.

By the term "mind," I mean ideas and purposes. By the term "body," I mean stuff and process. Stuff and process are familiar to every physicist as mass and energy in space and time, but ideas and purposes he keeps only in the realm of discourse and will not postulate them of the phenomena he observes. In this I agree with him. But what he observes is some sort of order or invariance in the flux of events. Every object he detects in the world is some sort of regularity. The existence of these objects is the first law of science. To detect regularities in the relations of objects and so construct theoretical physics requires the disciplines of logic and mathematics. In these fundamentally tautological endeavors we invent surprising regularities, complicated transformations which conserve whatever truth may lie in the propositions they transform. This is invariance, many steps removed from simple sensation but not essentially different. It is these regularities, or invariants, which I call ideas, whether they are theorems of great abstraction or qualities simply sensed. The reason for excluding them from physics is that they must not be supposed to be either stuff or process in the causal sequences of any part of the world. They are neither material nor efficient. So, to my mind Newton, Planck, and Jeans sin by introducing God as a sort of mind at large in the world to account for physical effects, like the action of gravity at a distance.

But let us now compel our physicist to account for himself as a part of the physical world. In all fairness, he must stick to his own rules and show in terms of mass, energy, space, and time how it comes about that he creates theoretical physics. He must then become a neurophysiologist (that is what happened to me), but in so doing he will be compelled to answer whether theoretical physics is something which he can discuss in terms of neurophysiology (and *that* is what happened to me). To answer "no" is to remain a physicist undefiled. To answer "yes" is to become a metaphysician—or so I am told.

But is that just? The physicist believes entropy to be somehow in or of physical systems. It may or must increase with time. But it is neither material nor efficient, in fact it is a number, namely, the logarithm of the probability of the state. It is, therefore, a measure of the disorder of an ensemble—or collection of systems. Now Norbert Wiener has proposed that information is orderliness and suggests that we measure it by negative entropy, the logarithm of the reciprocal of the probability of the state. Let us, for this argument, accept his suggestion. Ideas are then to be construed as information. Sensation becomes entropic coupling between us and the physical world, and our interchange of ideas, entropic coupling among ourselves. Our knowledge of the world, our conversation—yes, even our inventive thought—are then limited by the law that information may not increase on going through brains, or computing machines.

The attempt to quantify the information leads to a search for an appropriate unit, which, in turn, forces us to distinguish between two types of devices. In so-called logical, or digital, contrivances, a number to be represented is replaced by a number of things—as we may tally grain in a barn by dropping a pebble in a jug for each sheaf. The abacus is such a device. The nervous system is par excellence a logical machine. In so-called analogical contrivances a quantity of something, say a voltage or a distance, is replaced by a number of whatnots or, conversely, the quantity replaces the number. Sense organs and effectors are analogical. For example, the eye and the ear report the continuous variable of intensity by discrete impulses the logarithm of whose frequency approximates the intensity. But this process is carried to extremes in our appreciation of the world in pairs of opposites. As Alcmaeon, the first of experimental neurophysiologists, so well observed, "the majority of things human are two"—white-black, sweet-bitter, good-bad, great-small. Our sense organs, detecting regularities the same in all respects save one, create dichotomies and decide between opposites. These the "brain somehow fits together." From this sprang associationalism, culminating in Mill's evolutionary hypothesis that things are similar for us which have occurred together in the experience of our progenitors, and Kapper's law that nervous structures associated in action become associated in position. Neither proposes any mechanism other than random variation and the survival of those in whom the happy concatenation occurred. We inherit a nervous system so structured that we do perceive similarities (or have ideas) and these, not isolated, but conjoined within the system in many useful ways. That synthetic a priori is the theme of all our physiological psychology, learning excepted.

How, in all these processes, can we "quantify" the amount of information? In analogical devices it is best done by examining the numerical component. They all suffer from a peculiar limitation of accuracy which can usually not be pushed beyond one part in a thousand and almost never beyond one part in a million, even in such a simple matter as weighing. Moreover, analogical devices can not be combined in any way to push the decimal point. By measuring carefully the diameter and circumference of a circle, we might analogically estimate the ratio π to six significant figures. With a digital device, say an abacus, we can compute it to any number of places.

What characterizes a digital, or logical, device is that its possible states are separated sharply. In the simplest case there are only two. Wiener proposes that, for these bivalent systems, we define the unit of information as the decision *which* state it shall occupy. Notice, now, that this system has one degree of freedom—say, go or no-go—until it receives one unit of information, whereupon it has none. Next consider an ensemble of two such systems. It can be in any one of four states, and two units of information are required to match its degrees of freedom. If it were composed of three systems it might be in any one of eight states, and would require three units of information to fix it. Thus the number of possible states is 2 raised to the power which is the number of systems and each unit of information subtracts one from that exponent. Here Wiener's unit of information is exactly the logarithm to the base 2 of the reciprocal of the probability of the state, which, of course, is the negative entropy of an ensemble of bivalent systems. Neurons are bivalent systems.

Let me define "corruption" as the ratio of information in the input to that in the output. Each eye has something like a hundred million photoreceptors, each of which in a given millisecond can emit one or no impulse. In other words, it is an ensemble which can be in any one of $2^{100,000,000}$ possible states, or the amount of information it has is a hundred million units per millisecond.

Now Pitts and I have computed the information in the output of a piano player surpassing any ever known. We have given him a keyboard of a hundred keys, let him strike independently with each finger with any one of ten strengths ten times per second, and let each hand span ten keys. That sounds like a lot of information, but on computing it we find it is only about two units per millisecond.

Recent telephonic devices have sampled waves every thousandth of a second and passed on one pip if the wave was then of a given deviation from the mean, otherwise no pip. These are relayed to a smearing device and heard. It is better than 90 per cent as intelligible as the original voice. Three such pips per millisecond determined by eight possible values of the wave reproduce an orchestra. So much information at most may we hope to convey.

Whether we figure the ratio of input to output from our impossible player or from human speech, the corruption is of the order of a hundred million to one. Part of this corruption is referable to the coupling of our nervous system to our muscles and is avoided in some of the Crustacea. They use axons of several sizes, and by varying the frequency of discharge obtain more degrees of contraction than there are possible synchronous states of the nerves to their muscles. The viscosity of muscle smears the result in time, so that the rate at which impulses can come over the nerve is wasted by the inability of muscle to follow. In us a nerve of a thousand axons can be in 2^{1000} possible states, whereas the muscle, because it can only add tensions, has only a thousand possible states. 1000 is about 2^{10}; so the corruption in passing from nerve to brawn is 100 to 1.

What becomes of all the rest of the information? To answer that, conceive neurons as telegraphic relays. Each one may be tripped by some combination of signals provided these are very nearly synchronous. It detects the coincidence and only then emits a signal to subsequent relays. Now the threshold of the photoreceptors of the eye is always varying. At any one millisecond it may be tripped by a single photon, and, at another, fail to fire in response to many. By connecting many of these to a coincidence detector set to require a reasonable number of impulses simultaneously, we have a signal which corresponds to a statistically significant fraction of its receptors and so we wash out the random variation of threshold. Thus using the relayed information that fails to agree with other information, we achieve a high probability that what goes on through the nervous system does correspond to something in the world. Perhaps it will be clearer to say it this way. The logical probability that a neuron will have an impulse in one millisecond is 1/2, that two neurons of an ensemble in the same millisecond 1/2 × 1/2. The chance that both will fire by chance simultaneously is the product of their probabilities separately; that is, it is smaller; 1/4. Therefore, in the nervous system, by repeatedly demanding coincidence we vastly increase the probability that what is in the output corresponds to something in the input. We pay for certainty with information. The eye relays to the brain about the hundredth part of the information it receives. The chance that what it does relay is due to chance is fantastically small, 2^{-100}, a billionth of a billionth of a billionth of a tenth of one per cent.

Here, then, is the first technically important difference between us and robots. In them we cannot afford to carry out any computations, no matter how simple, in a hundred parallel paths and demand coincidence. Consequently, no computing machine is as likely to go right under conditions as various as those we undergo.

Accordingly to increase certainty every hypothesis should be of minimum logical, or a priori, probability so that, if it be confirmed in experiment, then it shall be because the world is so constructed. Unfortunately for those who quest absolute certainty, a hypothesis of zero logical probability is a contradiction and hence can never be confirmed. Its neurological equivalent would be a neuron that required infinite coincidence to trip it. This, in a finite world, is the same as though it had no afferents. It never fires.

In all of this I take it for granted that you are familiar with the all-or-none law of the nervous impulse, with the brevity of latent addition, with the duration of synaptic delay, with the evidence for spatial summation and for inhibition at a synapse, and with the local origin of energy from metabolism, all of which together insure that the principal circuit actions of the nervous system are those of the digital, or logical, kind. My reason for letting time flow in lapses of a millisecond is based on the work of Lorente de Nó, who has given us some of the best measures of the other properties of conduction. It is probable that no neuron can sustain more impulses than a thousand per second, even under the healthiest conditions, and one millisecond will include synaptic delay, or absolute refractory period, or the front of the pulse itself. These permit us to treat the nervous impulse as an atomic event.

But a nervous impulse is also a signal. It is true if what it proposes is true, otherwise it is false. It is false if it arises from any cause other than the adequate, or proper, excitation of the cell. The threshold of the dark-adapted eye for light is about a photon in several seconds. Pressure applied to the eye will evoke impulses, but the energy required is many million times more. Press on the eye and you see light when there is no light. The signals are false. Thus nervous impulses are atomic signals, or atomic propositions on the move. To them the calculus of propositions applies provided each is subscripted for the time of its occurrence and implication given a domain only in the past. In terms of such a calculus applied to nervous nets, Pitts and I have been able to prove that even nets devoid of circles can realize any proposition which is a logical consequence of its input. As this is the most that *any* net *can* do it is obviously an adequate theory. We know, of course, that facilitation and extinction occur, and we showed that whatever these can effect can be done digitally, or discretely, by go, no-go devices. In our first essay, we were unable to obtain much more than the calculus of atomic propositions; but, by introducing circles in which a train of impulses patterned after some fact could circulate, we did get existential operators for time past.

This is the argument: In a net in which there are no re-entrant paths a signal anywhere in the net implies a signal in a neuron nearer to receptors, and so backward in time until we arrive at the receptors. The signal here and now in this net implies the signal sent there just then. But once set going, a disturbance in a closed chain implies that there was a signal in its input at some time but does not indicate at what time. In short, the reverberating activity patterned after something that happened retains the form of the happening but loses track of when it happened. Thus it shows that there was some time at which such and such occurred. The "such and such" is the idea wrenched out of time.

It is an eternal idea in a transitory memory wherein the form exists only so long as the reverberation endures. When that ceases, the form is no longer anywhere. Only this kind of memory remains to aged brains in which no new abiding traces can be made and old ones fade. While we are young, use leaves some sort of change, as freshets cut their channels in the hills so that aftercoming waters follow and enlarge their beds. Yet all other forms of memory, including written records, do nothing which cannot also be achieved by mere reverberation, and hence add nothing to the theory.

There are other closed paths important in the origin of ideas, circuits which have "negative feedback." In terms of them reflexes were first defined as actions starting in some part of the body, setting up impulses to the central nervous system, whence they were reflected to those structures in which they arose, and there stopped or reversed the process that gave rise to them. All inverse feedbacks have this in common, that each establishes some particular state of the system, for they bring it back toward that state by an amount which increases with their deviation from that state. They are, as we say, error-operated. The state toward which they return the system is the goal, or aim, or end *in and of* the operation. This is what is meant by function. On these circuits Cannon founded his theory of homeostasis, and Rosenblueth and Wiener their theory of teleological mechanisms.

Any such circuit becomes a servomechanism as soon as the particular state it is to seek can be determined for it. Thus the stretch reflex tends to keep our muscles at constant length, but *that* length is determined for these circuits by more complicated arcs which traverse almost all parts of the central nervous system and require the reflex to seek those states which permit us to stand and move.

One reflex turns the eyes toward anything that enters the visual field. Its path runs from the eye by fibers that bypass the geniculate to enter the superior colliculi upon which they map the visual field. Here local circuits compute the vector from the center of gaze to the center of gravity of the apparition and send this information to the oculomotor nuclei which, in turn, relay orders to the appropriate muscles and turn the eyes so as to decrease that vector. As it reaches zero the eyes come to rest with the apparition centered. This reflex, I am told, will operate even in a man who has lost one-half his visual cortex, if he is dark adapted and a light, unseen by him, is placed in his blind field. If two are placed there the eyes turn toward a position intermediate. Under these conditions but with cortex *intact*, the eyes turn similarly but then snap from spot to spot, for the reflex is then subservient to impulses from the cerebrum. By turning the eyes so as to center the form, the reflex rids the apparition of the gratuitous particularity of the place at which it appeared. Every reflex, by running through a series of intermediate states to that established by it, rids some item to be observed of some fortuitous specificity. In the case of the collicular reflex, it has selected the centered form from among all possible exemplifications. Once in this, the canonical position, the system is ready for the computation of the form. There is little doubt that in us this computation occurs in the cerebral cortex, notably the visual areas. What happens then as the eyes turn rapidly is a series of on and off signals from most portions of the eye. These serve to clean the slate for the centered form unencumbered by blurring due to motion. The latter we suffer only when the eyes turn slowly; for instance, when we are very tired.

There are negative feedbacks within the brain. One of these resembles the automatic volume control in the radio. It tends to keep constant the sensory input to the cerebral cortex. In so doing it gives us another existential operator, for it detects that there was some intensity such that it was of this-or-that figure. In the case of vision, this circuit follows two other devices serving the same end; namely, the slow adaptation of the retina and the rapid change of pupillary diameter. All together these enable us to detect the form though the intensity of its illumination range through 39 decibels, that is, from faint starlight to full daylight—only we may not look at the sun without closing our eyes.

There are also appetitive circuits with a part of their path, from receptors to effectors, inside us, and the rest through the world outside. They are said to be inverse feedbacks over the target. Given any two inverse feedbacks which working together would destroy us, like swallowing and inhaling, there is built into us some connection between their paths whereby, when both are set going, one stops the other. In the case of learned modes of appetitive behavior, similar inhibitory links must be acquired or we perish. If we have three incompatible circuits in which *A* dominates *B* and *B* dominates *C*, the chances are equal whether *A* will dominate *C* or *C*, *A*. We speak of the end-in-operation of the dominant as of *greater* value. We have even tried to construct scales of value for diverse ends, but, since dominance is sometimes circular, values are not magnitudes of a single kind, and the terms "greater" and "less" are simply inapplicable. What we have called the value anomaly and regarded as evidence of a lack of order or system bespeaks, in fact, order of a kind we had not imagined, and a system tighter knit. Here endeth the psychological blind alley and Plato's theory of the Good. We cannot make one scale of value that predicts choice. Only knowledge of mechanism permits such prophecy.

I drew a circuit to move a figure, given anywhere in a mosaic of relays, to all positions in one direction. From each relay impulses ascended diagonally in the required direction through sheets of relays resembling the original mosaic and so spaced that their constituent relays formed columns perpendicular to the planes. And I set the threshold of all relays so that none would fire except when a slanting impulse coincided with one in the plane of that relay. I brought the output of every relay vertically all the way down to the original mosaic. Now when there is a simultaneous volley of the required figure at the given place in the original mosaic and at the same time a simultaneous volley to all the relays of any one of the sheets above, the figure is reproduced on that sheet by a volley in the relays where the slanting volley hits the sheet. Thence it projects straight down on the original mosaic. This reproduces the figure at a distance which steps off in the direction of the slant by a number of relays proportional to the height of the excited sheet. Now let the figure endure by a series of volleys at its origin, and excite the sheets successively upward, and the figure will be translated step by step from the origin to all possible positions in the direction of the slant. Whatever shape is present in the input to this circuit is preserved in these successive representations and, as the output descends vertically, the shape is translated without distortion. Von Bonin, who had worked with me on the auditory cortex, when he saw the diagram mistook it for a drawing of that cortex. Certainly we had but to replace the relays by pictures of neurons and the similarity was startling. The parallel functions are even more alike. We can center a form seen by turning our eyes, but there is no way we can tune our ears so as to translate a chord up and down the scale. Our brain receives it at a fixed key or pitch. In the primate these pitches map longitudinally on the input to Heschl's gyrus, so spaced that octaves span nearly equal distances. If this, the primary auditory cortex, worked like my circuit it would move the output up and down the axis of pitch while it preserved the interval, and so the chord. Here is an existential operator for chord regardless of pitch. The output asserts that there were pitches such that there was this or that chord.

Is there anything in physiology corresponding to the sequential excitation of the sheets? And, if so, how fast can it complete a cycle? There is the familiar alpha rhythm of the cortex, a shift of voltage that rises and falls through the cortex ten times per second. Although the correspondence *may* be entirely fortuitous, this is about the rate at which chords can be distinguished—ten per second. Now we need excellent histological studies by the Golgi method to know whether the detailed connections of cells in this area are what the hypothesis requires. These must be made in specified planes to match our physiological data. Because incoming signals and outgoing signals, like the pulse of scansion, ascend and descend through the cortex, when the cortex is at work the sweep of scansion should disappear, as it does, in the twinkle of details.

There are at least two ways that the output of this primary cortex may convey a chord regardless of pitch. There may be an inverse feedback which stops the figure of excitation when it reaches a canonical position along the axis of pitch, but there is no evidence that this exists. The other way is suggested by anatomy. Beneath the receptive layers of the cortex are columns of cells where properly timed impulses may be accumulated through a time equal to the sum of their synaptic delays to coincide upon efferent cells whose axons go to the adjacent, or secondary, auditory cortex.

If they terminate there at random, and if the cells there merely require coincidence to fire, we will have for every chord regardless of pitch a corresponding spot of maximal coincidence. The activity of this spot proposes the required universal, or idea. If we were to excite this spot electrically in waking man at operation, he should report hearing the chord. He does, but unfortunately no one has asked him whether he hears it at some particular pitch. The experiment is difficult because the primary auditory cortex is buried deep, and the secondary adjacent to it almost as deep, in the fissure of Sylvius. Moreover, I have not been able to map well the projection of the primary upon the secondary; and, finally, the interpretation is complicated by a direct projection upon the second with the sequence of pitches reversed. Fortunately these difficulties are not present in the visual area of man or monkey.

I drew a circuit to extract shape regardless of size, and this was mistaken by both von Bonin and Percival Bailey for a schematic representation of the outer strip of Baillarger which makes the visual cortex the "area striata." We start again with a mosaic. Select a point to represent the center of gaze and map the visual field as a set of concentric circles whose radii are proportional to the logarithm of the angles at the eye. From the mosaic let impulses proceed along branching channels spraying outward as they ascend through sheets of relays in which the density of relays decreases but their threshold increases as we go from below upward. From all of these relays let signals rise to corresponding upper layers of relays where coincidence with sweeping pulses is required, and let the signals of these layers converge on relays of low threshold, thence descend to leave the area striata. Now, with the pulse of scansion we shall have successively in this output all possible dilatations and constrictions of any figure in the input. The possibilities are limited by the grain and gross dimensions of the cortex, but these limit input and output equally. Since we have, in the output, all sizes made from a given one it makes no difference which size was given in the input.

Had we not conformed to present knowledge by mapping radial angles by their logarithms we would be compelled to require that the branching ascent of the input take a radial direction, but as it is it may branch nearly equally in all directions. Hence we cannot hope to detect much difference in histological study, even by silver stains of fibers, between sections cut radially and others, tangentially, in the visual cortex, or even in ones parallel to the surface. Thanks to Ramon y Cajal and Lorente de Nó, we know that the anatomical connections are at least sufficient for the theory. Here, as in audition, if the alpha rhythm evinces the scansion, we should be able to see ten forms per second. We can. Faster, they blur, merge, or glide into one another. Moreover, a rise of metabolic rate with fever or hyperthyroidism causes a rise in alpha frequency and a rise in the number of distinguishable frames per second.

When strychnine is applied locally to a spot on the cortex it causes the cells there to fire almost in unison. The fibers leaving that area then carry nearly synchronous volleys of impulses. These can be traced to the ends of the axons if there are enough of them near together. When these axons turn up again into the cortex anywhere, we can detect them there as a sharp change in voltage, the so-called "strychnine spike." When Dusser de Barenne and I strychninized a pinhead spot on the area striata, strychnine spikes appeared at many points in the secondary visual area as if the output from each spot in the area striata were scattered at random in the secondary visual area. Hence, from any particular set of spots in the primary area there will arise by chance some spot of maximum excitation in the secondary area. Activity at this spot implies activity in some figure of spots output by the primary; hence some shape regardless of size. Electrical stimulation of a spot on the primary visual cortex in waking man is reported by him as a blurred circle of light, whereas similar excitation of a spot in the secondary area is reported as a form. Moreover, this form, while it has a position in space, in the sense that he can point at it, has none in the visual field. Nor does it seem to have size there, any more than the recalled image of the moon seems to subtend one particular angle at the eye.

The mechanism we propose for abstracting chord and form is really computing a kind of average, and that average will not be seriously affected by small perturbations of excitation, of threshold, or even of particular connections as long as they are to cells in the right neighborhood. This conforms to clinical findings. A man may have several holes in his visual cortex, as big as or bigger than, the hole in his retina called the optic disk, and, except in a small number of cases, the forms seen will be unaffected. Although in such a case we can map these blind spots, he will not see them and the things seen will appear to be continuous through the blind spots. Scrutiny of the hypothesis even suggests that this process may account for much relational determination, for the four corners of a square in the input would be completed as a square, whereas parts of the sides might well flop, seeming now a maltese cross and now a square, etc. These flops would be the outcome of rivalry between two maxima for dominance over subsequent areas. Thus, for vision, our hypothesis fits well all known facts.

Older schools of physiological psychology and of neurology, guided by atomistic associational doctrines, tended to think in terms of neurons, each of which had one duty, for example, to know squares. This seems to be at least partially true of spots in the secondary visual cortex. Gestalt psychologists have treated the mosaic of relays of the cortex as if it were a field on which sensations mapped synchronously. This seems more likely true of receptors like the retina, for even its cortical replica is bisected by a line down the middle of the field and the halves mapped far asunder. Now it is easy to show that both of these "caricature" the nervous process. We need only note that a nervous net can take any figure in space, requiring an ensemble of a given number of neurons simultaneously, and convert it into a figure of impulses over a single neuron requiring as many relay times as there were neurons in the ensemble, and vice versa. From this alone it is clear that we cannot tell what kind of thing we must look for in a brain when it has an idea, except that it must be invariant under all those conditions in which that brain is having that idea. So far we have considered particular hypotheses of cortical function. They are almost certainly wrong at some point. Because they have already had to fit many disparate data, they are of little a priori probability. They prophesy the outcome of an infinite number of experiments, some of which are almost certain to refute them.

But with respect to the underlying theory, which is merely glorified tautology, there is no such possibility. It is, in fact, little more than a simple application of the theory of groups of transformations. For any figure in the input of a computing machine it is always possible to calculate an output invariant under a group of transformations. We calculate a set of averages, for all members of the group, of numerical values assigned by an arbitrary functional to each transform of the information conceived as the distribution of excitation at all points and times in an appropriate manifold. To define the figure completely under these transformations, we would need a whole manifold of such averages for various functionals, and this manifold would have to have as many dimensions as our original one; but, for practical purposes, we usually need only a few averages. Since in the finite net of relays the number of transformations in finite time is finite, we may use simple sums instead of averages.

This general theory describes all processes of securing invariants, or having ideas, which we have discovered or invented to date; and one mechanism differs from another in the nature of its arbitrary functional. For example, in the cerebral circuits proposed the functional may always assign the value one to any vector in the manifold if the particular point had a signal in the previous relay time, and, if not, assign it zero, whereas, in the reflex circuit for centering an apparition the functional clearly depends upon the figure of excitation in the manifold, and changes as the form centers; in effect, it assigns the value zero to all save the last transform on the cortex.

We may, of course, make the output of any calculator of invariants (or of several of them) the input to another and so have an idea of ideas, which is what Spinoza calls consciousness, and thus get far away from sensation. But our most remote abstractions are all ultimately reducible to primitive atomic propositions and the calculus of the lowest level. The domain of their implication lies only in time past. If their domain extended into the future, our sensations would imply our thoughts and our thoughts imply deeds. They do not, for even if the threshold of every cell in the nervous system were fixed, between the time we conceive an act and the time the impulses reach the motor horn cell, other signals from the world may get there first, and so often thwart us. We note the failure in the fact and are forced to distinguish between what we will and what we shall do. Hence the notion of the will.

But we do guess at things to come. When we run to catch a baseball we run not toward it but toward the place where it will be when we get there to grab it. This requires prediction. We behave as if there were some law compelling the world to act hereafter as it did of yore. Only one of our predictive circuits has been carefully studied by physiologists. It is responsible for optokinetic nystagmus. It has a tendency to persist, which may be seen when a train stops, for it then attributes motion in the opposite direction to the ties and rails. The earmark of every predictive circuit is that if it has operated long uniformly it will persist in activity, or overshoot; otherwise it could not project regularities from the known past upon the unknown future. This is what, as a scientist, I dread most, for as our memories become stored, we become creatures of our yesterdays—mere has-beens in a changing world. This leaves no room for learning.

Neurons are cheap and plentiful. If it cost a million dollars to beget a man, one neuron would not cost a mill. They operate with comparatively little energy. The heat generated raises the blood in passage about half a degree, and the flow is half a liter per minute, only a quarter of a kilogram calorie per minute for 10^{10}, that is, 10 billion neurons. Von Neumann would be happy to have their like for the same cost in his robots. His vacuum tubes can work a thousand times as fast as neurons, so he could match a human brain with 10 million tubes; but it would take Niagara Falls to supply the current and the Niagara River to carry away the heat. So he is limited to about the thousandth part of man's computer. He has to be very careful to specify in detail which relays are to be connected to a given relay to trip it. That is not the case in human brains. Wiener has calculated that the maximum amount of information our chromosomes can convey would fill one volume of the *Encyclopaedia Britannica*, which could specify all the connections of ten thousand neurons if that was all it had to do. As we have 10^{10} neurons, we can inherit only the general scheme of the structure of our brains. The rest must be left to chance. Chance includes experience which engenders learning. Ramon y Cajal suggested that learning was the growing of new connections.

I do not doubt that the cerebral cortex may be the most important place in primates. But it is certainly the most difficult place to look for change with use. Think of it as a laminated felt of fibers which serve to associate neighboring rough columns of cells nearly a hundred high and linked together vertically by their axons. These columns are then connected to distant columns by axons which dip into the white matter and emerge elsewhere into the cortex. These last connections I have studied for many years but have at best a general picture of how areas are related, certainly nothing that could give the detail necessary to distinguish between its connections before and after learning.

To understand its proper function we need to know what it computes. Its output is some function of its input. As yet we do not know, even for the simplest structure, what that function is. We have only a few input-output curves for the monosynaptic reflex arc obtained by David Lloyd, and now a few more by Arturo Rosenblueth. Walter Pitts is analyzing them mathematically at the present moment and has as yet no very simple answer. There is no chance that we can do even this for the entire cortex. That is why we need such a hypothesis as we have proposed for particular areas, for these may be disproved by records of electrical activity recorded concurrently at a few specified places.

Contrast our ignorance of its proper function with the detailed present knowledge of the projection of the sensory system upon it. For on at least two-thirds of its surface we can map the surface of the body, outside, and, to some extent, inside, so as to assign to every square millimeter of cortex the origin of its specific afferents and through them the exact position of the organs of sense. Beginning last summer, and continuing right now, the surface of the cerebellum, upon which the body maps similarly, is being stimulated and its projection to the so-called motor and sensory cortex, primary and secondary, explored and plotted millimeter by millimeter. Also now the projections of so-called non-specific afferents are receiving similar attention. Thus within a year or so we will know the geometry of its input and will be ready to seek in loci well defined the temporal pattern of its input.

I wish we could say half as much for our knowledge of its output. Since the days of Bubnoff and Heidenhain it has been electrically stimulated and the resultant change in muscle and gland carefully observed and elaborately recorded. But these responses depend upon the state of all subservient circuits which have yet to be analyzed. Hardly a month passes but what we are confronted by surprises. Frequency as well as shape of electrical pulses have been shown to determine the very path of the descending pulses from one and the same cortical focus. For example, volleys of impulses from the so-called face motor cortex, if more than ten or twelve per second, play principally through the nucleus of the seventh nerve upon the muscles of the face; whereas, if less than ten per second they are relayed almost exclusively through the nucleus of the twelfth to the tongue. Finally, the response to one and the same form of stimulation of a single focus in the motor cortex for one limb is determined both in amplitude and in direction by the motion and by the position of the limb at the time of stimulation. Clearly, to understand the significance of the output of the cerebral cortex we must know, for every subservient structure, the input-output curves. Even that will not be enough, for when several of them form a re-entrant circuit we must know their relations. Until we do so we will be in danger of attributing to the cerebral cortex functions proper to lower structures.

Last, but not least, the cortex is itself part of many re-entrant systems, and what our hypothesis attributes to cortex alone in securing invariants, or having ideas, may well depend upon loops joining it and the thalamus. From all these uncertainties I would turn to something simple as the monosynaptic arc of the stretch reflex and, by procedures far from normal, try to teach it something. It will be difficult, for in it the connections are as certainly determined as in a man-made computing machine; and we will have to break old connections before we can form new ones.

This brings us back to what I believe is the answer to the question: Why is the mind in the head? Because there, and only there, are hosts of possible connections to be formed as time and circumstance demand. Each new connection serves to set the stage for others yet to come and better fitted to adapt us to the world, for through the cortex pass the greatest inverse feedbacks whose function is the purposive life of the human intellect. The joy of creating ideals, new and eternal, in and of a world, old and temporal, robots have it not. For this my Mother bore me.

REFERENCES

1. Barker, S. H., and Gellhorn, E. Influence of suppressor areas on afferent impulses. *J. Neurophysiol.*, 1947, **10**, 125–132.
2. Bell, Charles. On the nervous circle which connects the voluntary muscles with the brain. *Proc. Roy. Soc.*, 1826, **2**, 266–267.
3. Cajal, Ramon y, S. *Histologie du système nerveux.* 2 vols. Paris: Maloine, 1909, 1911.
4. Lorente de Nó, R. Sections in J. F. Fulton's *Physiology of the nervous system.* London: Oxford University Press, 1943.
5. Maxwell, C. On governors. *Proc. Roy. Soc.*, '1868, **16**, 270–283.
6. McColl, H. *Servo-mechanisms.* New York: D. Van Nostrand Co., 1945.
7. McCulloch, W. S. A heterarchy of values determined by the topology of nerve nets. *Bull. of Math. Biophys.*, 1945, **7**, 89–93.
8. McCulloch, W. S. *Finality and form.* Fifteenth James Arthus Lecture, New York Academy of Science, May 2, 1946.
9. McCulloch, W. S. *Machines that think and want.* Lecture at the American Psychological Association, September 9, 1947.
10. McCulloch, W. S. *Through the den of the metaphysician.* Lecture at the University of Virginia, March 23, 1948.
11. McCulloch, W. S. Teleological mechanisms. *Ann. New York Acad. Sci.*, 1948, **50**, 4.
12. McCulloch, W. S., and Lettvin, J. Y. Somatic functions of the central nervous system. *Ann. Rev. Physiol.*, 1948, **10**, 117–132.
13. McCulloch, W. S., and Pitts, W. How we know universals. *Bull. Math. Biophys.*, 1947, **9**, 127–147.
14. McCulloch, W. S., and Pitts, W. The statistical organization of nervous activity. *J. Amer. Statistical Assoc.*, 1948, **4**, 91–99.
15. Rosenblueth, A., Wiener, N., and Bigelow, J. Behavior, purpose, and teleology. *Philos. of Science*, 1943, **10**, 18–24.
16. Wiener, N. *Cybernetics.* New York: John Wiley and Sons, 1948.
17. Wittgenstein, L. *Tractus Logico-Philosophicus.* London: Paul, 1922.

DISCUSSION

DR. LORENTE DE NÓ: The main question in our minds is whether the theory as a whole is going to stand or not. I think that probably many of the details will not stand, but that the main concept will certainly remain. I'm quite sure that all of my colleagues will agree that Dr. McCulloch has brought what we know of both the anatomy and the physiology of the brain closer to an integrated whole than it has ever been before, and I want to congratulate Dr. McCulloch very much and very sincerely.

DR. VON NEUMANN: I would not like to attempt a detailed discussion of the very beautiful and very interesting presentation made by Dr. McCulloch, perhaps something like that can be done in the general discussion. I will, however, ask two questions, both dealing with only one aspect of the matter. You have emphasized that you are giving *sufficient* mechanisms and that it is in conflict with your entire philosophy at this time to claim that these are necessarily the ones that are used. You give proofs of possibility. There is, nevertheless, one point where the question of the actual mechanism is especially burning, and that is the question of memory. You have pointed out that there are positive feedbacks—reverberating circuits—built out of switching organs which are quite adequate as memory. If there were nothing else in the world except neurons, you could build memory out of neurons. My own feeling is that if one were really to construct in this way a nervous system with its known attributes it would probably take more neurons than there are, but this is an aside. My real question is this: First of all, I have observed that all neurologists seem very certain that the reverberating circuit trick is not used in making the actual memory. Amorphous intuition points in the same direction. In surmising this is not so, I have always had a bad conscience. I am not sure why they are so positive. What is the best evidence one can give for this?

The second question is this. Most neurologists with whom I have had an opportunity to talk seem to be equally convinced that memory is due to some lasting changes somewhere on the body of the nerve cell, somehow connected with alterations of thresholds. Is it not better to say that there probably is a memory organ somewhere, but that we are absolutely ignorant as to where it is—probably as ignorant as the Greeks, who located the whole intelligence in the diaphragm?

DR. MC CULLOCH: I'm afraid my answer is necessarily a bit lengthy. In the first place, I would like to contrast as sharply as possible the maximum length of what I consider reverberative memory, with the enduring memories which we bring on from childhood. I have seen a man over 80 years of age walk into a meeting of a Board of Directors and for 8 hours work out from scratch all of the details necessary for the sale of a complete railroad. He pushed the other men so as to get every piece of evidence on the table. His judgment was remarkably solid. The amount of detail involved in the transaction was enormous, and it actually took over 6 hours to get all of the requisite details on the map. He summarized that detail at the end of the meeting, in a period of a half an hour, very brilliantly, and when he came out he sat down, answered two letters that were on his desk, turned to his secretary, and said, "I have a feeling that I should have gone to a Board of Directors Meeting." He was not then, or at any later time, able to recall one iota of that meeting, and he was in that state for nearly a year before he died. This is the picture of what we call "presbyophrenia." In that state, whatever our memory organ is, we are unable to make any new record in it. Actually, the recent paths begin to fade, leaving only earlier memories to pop up. So, at that period, one is more likely to remember in detail and individually, the things that happened in childhood rather than the things of later years. Such a memory goes, quits, stops, the minute the brain is used for something else, or the minute that it comes to rest. Here is a span of at least 8 hours of high cerebral activity in carrying the details from the first moment of the meeting to the end of the meeting.

Per contra, not all memory can be of this reverberative kind. It is obvious that, although this kind of memory is carried reverberatively in the brain, it cannot endure during very deep sleep and it cannot endure during narcosis. It goes out, when the brain has a seizure and it goes out in sleep. In the one case, it goes out because the whole apparatus is pervaded by what I will call shock waves which go through it and through it and through it in the fit. In the other case, it goes out because it has no signals traveling—the brain is "shut down." I believe that only lower mechanisms are really busy in deep sleep. Now then, why do we want to attribute the memory to the brain at all? Why may it not be in the spleen or somewhere else? The answer is because injuries of the brain, but not injuries of other things, do result in losses of memory, and that is the fundamental reason for pinning it on the brain.

The next question is: Why does one attribute this, the enduring memory, to a growth process—change with use—somewhere in the excitability in neurons rather than elsewhere? Well, first, because it is a relatively lively process, and when things are growing, one tries to pin it on growth processes. Second, because it has the peculiarity that what we learn later is only a modification of what is already laid down. It is an accumulative affair of this sort. Why attribute it to the junctions of cells? Because there is where we imagine the switching takes place, and this is the kind of evidence on which we base it.

Let me tell a tale out of school, even if future evidence fails to support it. I will ask Dr. Lettvin's forgiveness later. The theory and experiments are his, although I have done some of them with him. His theory, and experiments, are designed to meet the requirements of the conditioned-reflexologists, Pavlov et al. In deference to them we will name one source of afferents *U*, the unconditioned afferents, which can excite an efferent *R*, the responsive motoneurons, and a second set of afferents *C*, the conditioned afferents, and—Heinrich Klüver and Warren McCulloch to the contrary notwithstanding—we will forget for the moment "stimulus equivalence" or "universals secured by averaging over groups of transformation" and treat *U*, *C*, and *R* as individual neurons. *U* can always fire *R;* but *C* can become able to do so reflexologically only if *C* and *U* are excited so as to be active concurrently. I mean that if both are concurrently active then, thereafter, C alone shall be able to fire R. Now the gist of Dr. Lettvin's analysis is that it still further simplifies the required assumption. He asks, for what do we need *U*—except to excite *R*—so why not make the simple assumption that if *C* and *R* are simultaneously active, *C* shall become able to fire *R*? Naturally this simplification should not occur to a psychologist for he has to use *U* to excite *R*. But a physiologist may put his electrodes directly upon *R*, if he can get them there, or he may fire it antidromically.

Now it is clearly established by the surgically and electrically perfect experiments of Donald Marquis and Arthur Ward that the intact spinal cord cannot be conditioned. But Culler and Shurrager did sometimes obtain conditioning of what they believed to be the two-neuron reflex arc, and this in experiments on by no means their technically best preparations. Wiener's theory that the spinal cord has suffered a "Wärmetod" of information by the time we are able to walk (that is, its connections are all soldered in) would account for this discrepancy. One has only to suppose that one must destroy something ending on a motoneuron to leave root room for another afferent; and the cord, no longer intact, could be conditioned even as Cajal supposed, by something else making connection with the motoneuron.

Moreover, the technically impeccable experiments of David Lloyd have proved that of two muscle antagonists at a single joint, each by its afferents (from stretch receptors) inhibits, and only inhibits, its antagonist. They show further that this inhibition occurs at the synapse on the motoneuron without time for internuncial intervention.

It follows, that if one were to cut the dorsal roots of the nerves for extension at the knee, there would be root room on its motoneurons for afferents from the flexors of the knee to get a greater hold on these motoneurons. And Dr. Lettvin's ingenious theory of synaptic transmission predicts that, if this happens, instead of inhibiting the motoneuron, these afferents will then excite them.

This, in substance, is what we did. We cut the dorsal roots of the extension reflex, stimulated its ventral roots antidromically, and at the same time stimulated the flexor muscle nerve. This we did to both for a long time, minutes or even hours, at about forty per second, from separate stimulators. Thereafter, but not before, threshold stimulation of the flexor nerve elicited contractions of the extensor. Thus Dr. Lettvin has proved that the cord, no longer intact, can be conditioned, but not quite as the psychologist would have it—for he stimulated *C* and *R*, not *C* and *U*, concurrently. He has not as yet published, and will not publish, these findings to physiologists until his records of the times of these impulses at dorsal and ventral roots show conclusively whether or not this functionally new path is monosynaptic.

If we are not misled by the sensitivity of denervated structures, and the cord does so learn, this is crucial to psychologists. The cord is a sufficiently simple structure and is sufficiently well known for us to hope that an anatomist, with some new technique, may be able to find structural changes.

From what I have said, it should be clear that I do not think learning normally occurs in the spinal cord. Even in the earthworm learning seems to reside, albeit not in the most anterior segments, still in the forward ganglia. In mammals it may be in the midbrain, or even in the cortex, but our chances of locating anatomical changes there are negligible.

DR. VON NEUMANN: The experiment which you described—if it were done, and if the time relations were clear—would be very convincing. In this case one could at least feel certain that the "conditioning" consists in a physical change in the cell actually under consideration.

The "reverberating circuit" model for the memory does not strike me, as I said before, as a particularly elegant one. Nevertheless, it is important to know whether it is a possible model or not. I understood you to state that it is not. What is the decisive argument against it? I understood it to be that there are states when one can be fairly certain that the cortex is totally inactive and yet memory persists. What exactly is the evidence for this "total inactivity" of the cortex? Is it that one has not so far succeeded in picking up any electrical signals from it?

DR. MC CULLOCH: That's right. When our amplifier is turned up maximally, we pick up activity only from the respiratory mechanism and similar structures. Only *they* keep on going in deep sleep and in the coma following seizures.

DR. VON NEUMANN: Is this reliable enough to know that there is nothing else there?

DR. MC CULLOCH: Well, let's take the more powerful case—that in which you have a seizure with tremendous waves of signals through the works.

DR. VON NEUMANN: Does one know that they are really going through all channels?

DR. MC CULLOCH: I think, pretty firmly, yes; I don't believe any part of the nervous system is unaffected.

DR. VON NEUMANN: The organism is fairly well set up to protect certain parts, is it not?

DR. MC CULLOCH: The grand mal convulsion is the occasion on which that protection breaks down.

DR. VON NEUMANN: It does not seem to for memory.

DR. LORENTE DE NÓ: The difficulty in making memory reverberating paths is chiefly this: To maintain a steady state in any kind of reverberating path, the closed chains of neurons are arranged so that either you have incremental activity or decremental activity. Either the thing begins spreading to involve more and more and more neurons, or it decrements, after coming to a maximum, and then decays and disappears. Probably the secrets that Dr. McCulloch just gave us can be compared with the gramophone record. While we are playing a gramophone record, we have an articulation of impulses; later, when the record is over, memory is deposited in a different manner. As you listen to what I am saying now there are a lot of circuits operating according to those principles; but, then, when in a moment I stop talking, those signals will have stopped and memory will be somewhere else, in some other area.

DR. VON NEUMANN: I see the plausibility of what you say, but I still have a residue of uncertainty left. Your arguments about electrical circuit analogies are plausible, but they are nevertheless influenced by our particular kind of experience in this field. Your judgment based on anatomical experience is perhaps more cogent. It may be anatomically established that closed (and hence potentially reverberating) neural pathways do not exist in the necessary, vast numbers.

Another comment I would like to make is this. I see an argument that one might make against the view that memory in any form actually resides in the neurons. It is a negative argument, and far from cogent. How reasonable is it? This is the argument: There is a good deal of evidence that memory is static, unerasable, resulting from an irreversible change. (This is of course the very opposite of a "reverberating," dynamic, erasable memory.) Isn't there some physical evidence for this? If this is correct, then no memory, once acquired, can be truly forgotten. Once a memory-storage place is occupied, it is occupied forever, the memory capacity that it represents is lost; it will never be possible to store anything else there. What appears as forgetting is then not true forgetting, but merely the removal of that particular memory-storage region from a condition of rapid and easy availability to one of lower availability. It is not like the destruction of a system of files, but rather like the removal of a filing cabinet into the cellar. Indeed, this process in many cases seems to be reversible. Various situations may bring the "filing cabinet" up from the "cellar" and make it rapidly and easily available again. There are many examples of this: the "forgetting" and subsequent "remembering" or recovering of languages, telephone numbers, names—paralleling the decreased or increased need for their use.

This organizational situation is a very plausible one, if there is a memory which is much larger than the available switching facilities for its selective use. Indeed, if the memory is thus larger than its switching system, it will be necessary to introduce a system of priorities for various parts of the memory. Each part may then, upon occasion, be moved into regions with rapid accessibility, or into regions with less rapid accessibility. Or, rather, it may not be moved from region to region, but be connected to quickly or to less quickly functioning portions of the available switching system.

If this is so, then the memory cannot reside in the actual switching organs in the neurons, and its capacity must be much greater than that represented by the switching system. One must then postulate a very high-capacity memory organ or organization, with considerable bottlenecks at the "input" and "output," that is, at the points of contact represented by the switching system.

Does this sound plausible, or is there some flaw in my argument?

DR. BROSIN: May I break here with the tradition of immediate reply? This is a very large subject, and I would like to see if there are other commentators. You may gather the evidence, and if we do not have time enough today, you can have a full dress performance tomorrow.

DR. GERARD: I would like to ask a few questions, some of which have already been touched upon. I have a very trivial one first. I didn't quite see why you place such disparate emphasis on the manipulation of the output of the brain in efferent systems and paths, as compared to the problem of the manipulation of the input of the brain. If I correctly understood you, you are not particularly worried about the input side. I don't see why that is, and I would like to have you explain it a little more clearly.

I have, also, two other points that touch on this memory problem. If learning and remembering are based on growth processes of some sort, then they should not be basically different from developmental and maturational behavior; and yet it seems to me that some of the most striking experimental work in the past does emphasize a very fundamental difference between the maturational learning in the nervous system and acquiring a new behavioral capacity—experiential acquiring of new behavior possibilities. The former takes place certainly without any external experience, but you can see that there is internal experience. On the question of rest and activity of the nervous system, several members of the audience, during our intermission, raised the question with me whether you are not neglecting something that you might call automatic activity of neurons. The assumption is that the output will be determined by the input, rather than by something happening independently of the input. I will put the question to you in this way. Do I correctly assume that you were suggesting that the scansion machine in your projection area mechanism is the spontaneous brain wave, and whether it starts there or below is immaterial? I would like to have that elaborated and made a little bit clearer. I personally am surprised at the answer you gave Dr. von Neumann, that the brain can be completely quiet. I don't believe that electrically, or in any other way, it is ever completely quiet at any time except in death.

DR. MC CULLOCH: I said, except for lower mechanisms.

DR. GERARD: I believe that, even in the other mechanisms, I have never seen a completely silent brain.

DR. MC CULLOCH: No, I don't believe that brain matter is ever completely quiet. I'll take care of that question later. For the moment it is enough that there are times when no signals are reverberating.

DR. GERARD: If there is a separate memory organ, along the line of Dr. von Neumann's comment, in which you have your files easily accessible or down in the basement, that would argue against the memory traces being associated with the neurons themselves throughout the brain. What about the reversible amnesia problem, where all past memory vanishes for long periods and then comes back again? If learning involves the establishment of new functionally effective connections between neurons in the brain (whether by growth, by physiological change in threshold, or what not), and if that depends on activation of neurons and association with experience, then it seems to me that it should follow that if the threshold of neurons is held low, just in general during the experiencing of experience, learning should be enhanced. Dr. Lettvin raised the level of excitability of neurons in the nervous system. There is more chance of a particular input leaving a permanent modification, or even a temporary one. I hope some of the psychologists here can bring the evidence in, but I don't know. However, as far as I'm aware, conditioning under the influence of stimulating drugs has not changed the rate of this conditioning.

The last question I should really leave for Dr. Lashley, since it is in his field. If these networks of neurons (even allowing for considerable interchangeability of particular elements of the net) are organized so beautifully in the striate and elsewhere for these particular functions, then how do you account for some of Dr. Lashley's critical experiments on destruction of different parts of the brain and the retention of learning, memory, and all the rest of it?

DR. KÖHLER: I admire the courage with which Dr. McCulloch tries to relate his neurophysiology to facts in psychology. But, when in a skeptical mood, I sometimes feel like criticizing the results. Take the example of visual shapes which, as we all know, are generally recognized in a peripheral position (or in a larger size), even if, heretofore, they have been seen only in foveal projection (or in a smaller size). Dr. McCulloch's explanation of such achievements introduces more histological assumptions ad hoc than seem compatible with usual standards of plausibility. In fact, he does not seem himself to maintain that a real brain functions in this fashion. Why then the elaborate constructions? Most probably the reason is that the atomistic character of Dr. McCulloch's neurophysiology prevents any direct approach to relationally determined facts such as visual shapes. The difficulty seems to be strongly felt, and special sets of neuron connections are now being constructed which merely serve to remove the difficulties caused by the main atomistic premise. Would it not be simpler never to make this atomistic assumption? If we think of cortical function in terms of continuous field physics rather than of impulses in neurons, the difficulty never arises. The contours of retinal images are projected upon the visual cortex by nerve impulses. Let us assume that here they constitute the boundary conditions of field processes such as electric currents. Under these circumstances, there will be for each set of boundary conditions, that is, for each shape, a particular distribution of a directly interrelated function; in other words, each shape will be cortically represented by a specific process. If the characteristics of such a process remain approximately constant, independently of its location and size, then recognition of a shape in a new place or size offers no problem which is not also present in the recognition of a color in a new place.

Incidentally, it seems to me misleading to assume that the present problem is mainly a problem of recognition, and therefore of memory.

When two objects are given simultaneously in different places while the eyes do not move, we can compare these objects, and say whether they have the same shape. Once more the implication is that visual shapes are associated with specific processes.

Occasionally, I am afraid, Dr. McCulloch uses psychological terms in a strangely diluted sense. In fact, sometimes little is left of what they actually mean in psychology. But the change is never mentioned. People will therefore tend to believe that, when such terms are now being related to neurophysiological hypotheses, it is their real psychological contents which are given a physiological interpretation. They will not notice that the essential characteristics of the facts in question are tacitly being ignored. I have an uneasy feeling that this may happen even to the theorist himself. Thus Dr. McCulloch likes to call a nerve impulse a "proposition." Moreover, he says that the occurrence of a given nerve impulse "implies" the occurrence of preceding impulses (in other neurons), by which the given impulse has been started. But, typically, a proposition is concerned with a relation between certain terms, whatever the relation may be in individual instances. A cortical situation would therefore correspond to a proposition if in this situation the cortical counterparts of two terms were functionally related in one specific fashion or another. A nerve impulse does not in this sense relate two terms to each other. At least in Dr. McCulloch's neurophysiology, a nerve impulse seems to be a particularly lonely event. How, then, can a nerve impulse represent a proposition? Some discussions of nerve impulses and of their equivalence to facts in psychology make me feel that, inadvertently, an extremely learned histologist and neurophysiologist is tacitly supposed to watch the human brain continually, and that this expert always knows how impulses must be interpreted in psychological terms. He probably tells the owner of the brain what psychological facts he must have when impulses travel in this or that part of the cortical machine. For without this help, what could induce a person to think of a specific proposition, that is, a particular relation between two terms, when an impulse travels in a certain fiber? Since, actually, no such expert is available, the characteristic forms of the various psychological facts must be directly given by the functional characteristics of corresponding cortical processes. But, to repeat, if this is the case, it cannot be nerve impulses which give propositions their relational character. For they have no such character themselves. For the same reason, there can be no connection between the psychological experience that one fact implies another fact and the behavior of a nerve impulse. A present impulse implies, say, preceding impulses in other neurons

(McCulloch's example) only in the mind of a neurophysiologist who knows what must have happened a moment ago at a certain synapse. As the present impulse travels along its fiber, it knows nothing of preceding impulses.

For a moment, I must come back to a criticism to which I have referred once before. It must be a hard task to give psychological facts interpretations in terms of nerve impulses. For when this task arises, and is apparently accepted, the theorists soon forget what they must now be expected to do, and turn to other problems which are only indirectly connected with the original problems. Invariably, such substitute problems are more accessible to explanations in terms of nerve impulses. On the other hand, since they are somehow related to the problems which were actually to be solved, psychological concepts which are essential in the latter will naturally also be mentioned when the substitutes are being discussed. Thus, if interpretations in terms of nerve impulses seem to work in the case of the substitutes, both the theorists and others will easily believe that actually the original problems have been solved. For this is what the theorists had promised to achieve.

Take "having a goal" as an example. Before we realize what is happening, the task of explaining this psychological fact in terms of nerve impulses has been replaced by another task: Once a person has a goal, how is the goal actually reached? Naturally, if this is done by overt action, both centrifugal and centripetal nerve impulses will play an important role in the process. It is also a most sensible suggestion that the action is steered in the right direction by negative feedback. But do we learn in this fashion what "having a goal" is in terms of nerve impulses? Plainly, we do not. Nonetheless, we may be so strongly impressed by what seems to have been achieved that we forget what had to be achieved. Of course, it must be difficult to understand "having a goal" as a matter of nerve impulses. "Having a goal" is again a relational situation. When a person has a goal, his self (in a purely empirical sense) is dynamically related to a certain object, and therefore, probably, the neural counterpart of the self to the counterpart of the object. Moreover, the nature of the relation depends entirely upon the perceived characteristics of the object and the state of the self at the given time. The theorists themselves seem to doubt whether interrelations of this kind can be mediated by nerve impulses, which are described as atomic events par excellence. Otherwise, why should the theorists prefer to discuss something else, namely, goal-directed action? And yet, "having a goal" is a problem which must be handled quite apart from overt action in reaching the goal. For people often have goals while they do not yet know how these goals can possibly be reached. It also seems probable that a really adequate interpretation of "reaching a goal" presupposes a correct interpretation of "having a goal." As a goal is being reached, the dynamic relation between the self and the goal, which seems to represent a store of energy, is gradually being changed—until eventually, when the goal has been reached, this energy is spent. I have a suspicion that the negative feedback involved in the change refers to the store of energy implicit in "having a goal." But, of course, this again is thinking in terms of field physics.

I will remark only in passing that the substitution of one problem for another occurs also in Dr. McCulloch's treatment of "value." He does not give us a theory of value in terms of nerve impulses. But values may conflict, just as many other things may conflict, and then the question arises which value will win in a given conflict; that is, which is the stronger value. It is this question with which he prefers to deal. But since the same question may be asked with regard to many facts which are not at all values, we have obviously once more lost our way. We may easily believe that we are actually dealing with the problem of value as such; and this belief will be strengthened by the fact that, in formulating our new problem, we may still mention the concept "value." But the problem what value means in terms of nerve impulses has in the meantime been forgotten.

Quite probably, Dr. McCulloch will not be impressed by these arguments. He may feel that I am accepting certain premises of which he does not approve. First of all, he is likely to say that the structural characteristics of cortical processes need not agree with the structures of corresponding psychological facts. Actually, he has just told us that the hypothetical cortical counterpart of an idea must fulfill only one condition: It must always occur when the idea occurs. More specifically, he has once said that the cortical counterpart of a square may be a "four-spoked form, not at all like a square." I cannot agree with this statement for the following reason. It would not be difficult to give subjects a series of tests in which they would have to respond to one structural characteristic of a square after another in overt action. Under these circumstances, the form of their actions would directly follow from the corresponding structural properties of the square. Their actions would prove, for instance, that their square has four straight sides, that pairs of these sides are parallel, that the angles have all the same size, and so forth. From the point of view of natural science, how can this happen if the cortical counterpart of the square has no corresponding characteristics? Does Dr. McCulloch suggest that such characteristics exist only in the square as an "apparition" (his term), that is, as a mental fact, and that, quite apart from the cortical situation, this mental fact as such determines what the subjects are doing? There is an old name for this view. It is called dualism. I find it hard to believe that dualism appeals to Dr. McCulloch. But in this connection he does argue as though he were a dualist.

If we consider how the visual square (the apparition) comes into existence, we meet with the same difficulty. How can a cortical process such as that of a square give rise to an apparition with certain structural characteristics, if these characteristics are not present in the process itself? According to Dr. McCulloch, this is actually the case. But if we follow the example of physics, we shall hesitate to accept his view. In physics, the structural characteristics of a state of affairs are given by the structural properties of the factors which determine that state of affairs. The magnetic field around a long conductor with circular cross section obviously describes circles; the electrostatic field around a charged sphere is symmetrical with regard to the center of the sphere, and so forth. Situations in physics which depend upon the spatial distribution of given conditions never have more, and more specific, structural characteristics than are contained in the conditions. To be sure, this rule holds only so long as the medium in which a physical situation develops is homogeneous, that is, devoid of special conditions of its own. For instance, the field around a charged sphere will no longer be symmetrical about the center if the environment contains various dielectrics in an arbitrary arrangement.

If we apply this lesson to the way in which the cortical counterpart of a square gives rise to this square, we must choose between two possibilities. Either the structural characteristics of the visual square are fully determined by its cortical counterpart. Then this cortical process must have the structural characteristics of the square. Or we assume that the visual square has structural characteristics of its own which are not present in the cortical process. Then the world of apparitions, the psychological world, constitutes a particular medium with special determining conditions, quite apart from cortical conditions; and it is these conditions in the mental world which add the structural characteristics not contained in the cortical process. The second alternative is, of course, again tantamount to dualism. It seems that if we do not want to be dualists we must accept psychophysical isomorphism.

DR. BROSIN: May I again beg your indulgence and give you a full opportunity to continue later? Dr. Lashley.

DR. LASHLEY: I am very much in sympathy with the type of development represented in the last two papers. I think any understanding of the nervous system we may acquire must be developed within the framework of our knowledge of the activities of the individual neuron. There may be additional factors introduced by combinations of which we know little or nothing at present, but the general principles seem to me to be fundamentally correct. At the present time, however, such a formulation involves a very great oversimplification of the problems. The behavior which is explained is behavior which never occurs in the intact organism. It is an hypothetical behavior derived from the assumptions of the system rather than a description of observed phenomena. A visual object maintains its continuity in spite of constant fluctuations in the position of the eyes and shifts in its position on the retina. By a series of special assumptions concerning neural organization this phenomenon of stimulus equivalence can be accounted for in terms of impulse switching. But in recognition of the visual object it makes little difference whether the image of the whole object falls upon the retina. If only part is seen at any one time, the entire form is rapidly reconstructed from the series of images of parts. The temporal sequence of part figures is combined with the spatial orientation of eye movements to give spatial continuity to the whole. This phenomenon requires a new set of assumptions to make the theory of impulse switching applicable. I somewhat question the utility of a theory which has to be revised to fit each special case.

In its present form the theory of impulse switching involves, I believe, assumptions concerning the accuracy and uniformity of neuronic structure which are not justified by the facts. We have been studying individual variations in the number and arrangement of neurons in the cerebral cortex. We find a wide range of individual differences in cell number and size in corresponding areas of different brains of animals which are grossly indistinguishable in behavior. Two brains may differ by as much as 50 per cent in the number of neurons in the temporal lobe or by 100 per cent in the average size of cells in the superior frontal convolution. A given type of cell may be present or absent from the auditory cortex or operculum. Yet the fundamental behavioral activities of these animals are the same. The anatomic variability is so great as to preclude, I believe, any theory which assumes regularity and precision of anatomic arrangement. Such facts lead me to believe that theories of neuron interaction must be couched, not in terms of the activity of individual cells but in terms of mass relations among the cells. Even the simplest bit of behavior requires the integrated action of millions of neurons; the activity of any single neuron can have little influence on the whole, just as the path of an individual molecule of a gas has little influence on the gas pressure. It is questionable whether specific instances of behavior can ever be dealt with in terms of the activity of individual neurons; the complexity is too great. We shall probably have to use a different kind of model, a model which can be explained in principle by individual neuron action but which involves a somewhat different set of concepts and laws of action. These laws may eventually be derived from study of the individual neuron when those properties are directly observed. At present, however, many of the properties ascribed to the individual neuron are inferred from the activities of neuron masses, and explanations based on such inferred properties are circular and, perhaps, spurious.

Some of the specific hypotheses which have been formulated by Dr. McCulloch seem to me to meet with serious difficulties. He has suggested a reverberatory system between the striate cortex, the suppressor band of area 19, the prestriate region, and the thalamus. I have just removed the prestriate region (including areas 18 and 19) from a series of monkeys and also the frontal eye fields (another suppressor area) singly and in combination. I have been able to detect no visual disturbances whatever following the operations. In no case have we been able to detect significant perceptual disturbances after removal of suppressor areas or of supposed sensory associative areas in monkeys. The specific hypotheses which Dr. McCulloch has suggested for the action of the visual and auditory analyzers imply a definite spatial position of the analyzing mechanisms. Experimentally they are not there.

This leads to the general problem raised by most of the experimental studies of effects of cerebral lesions. Limited lesions or interruptions of transcortical connections produce few or no symptoms. Behavior seems not to depend upon any localized conducting pathways within the cortex. Habits are not stored in any limited area. Such facts point to the conclusion that there is multiple representation of every function. I see no other way of meeting this difficulty except by assuming some sort of reduplicated network of equivalent functional circuits. In other words, we cannot deal with individual conditioned reflex arcs but only with a multiplicity of interacting circuits whose excitatory effects can be transmitted around various types of cortical interruption.

One other point, in relation to the problems of memory raised by Dr. von Neumann: For memory there is the same problem of equivalence as for transneural conduction. I have found, for example, that one sixtieth of the visual cortex of the rat will mediate visual memories and it may be any sixtieth, provided it includes part of the central projection field. Here, again, there must be some sort of multiple representation. The memory is not stored in a single locus.

Now consider the nature of a memory. It is not a single item which can be filed in a single neuron or reverberatory circuit. It is always the capacity to reproduce a series of events, to reproduce a complex sensory pattern or a series of motor activities. Such neural events involve the activity of millions of cells. I have come to believe that almost every nerve cell in the cerebral cortex may be excited in every activity. I shall give some quantitative evidence of this tomorrow. Differential behavior is determined by the combinations of cells acting together rather than by cells which participate only in particular bits of behavior. The same neurons which maintain the memory traces and participate in the revival of a memory are also involved, in different combinations, in thousands of other memories and acts. The memory trace is the capacity of many neurons to work together in certain permutations. In a system of interconnected neurons the number of possible permutations may greatly exceed the number of switching mechanisms. Perhaps this answers Dr. von Neumann's difficulty with regard to number of elements. It is also an argument against the dynamic as opposed to static character of the memory trace.

DR. BROSIN: Dr. Weiss, have you anything to add?

DR. WEISS: Much of my comment had better be left for a later part of the symposium. For the present I want to point out that we are actually dealing here with two different problems. Namely, first a statistical consideration, as it were, of whether or not the number of elements present in the nervous system and their various interrelations is sufficient to account for the number and variety of things it can do. We realize, and this gives us intellectual comfort, that the number of possible constellations is large enough to allow for the observed variety of behavior. This statement, to quote McCulloch, would be merely tautological. As a biologist, I am more interested in the second problem, and that is the precise pathways and chains of processes through which, out of the infinite variety of possibilities, just the appropriate sequence and selection are activated which lead to a given appropriate organized response. And if we deal with these mechanisms not as abstract categories, but in concrete terms, then I see some serious and realistic difficulties arising for any theory of nervous networks that requires the amount of precision postulated in the schemes here presented. The study of the developed nervous system, with which the anatomists, physiologists, and psychologists are usually working, suggests a high degree of precision in the arrangement of the constituent elements, but it must be realized that this impression is illusory. The organizational stability of a performance of the nervous system is much greater than the precision of the underlying structural apparatus.

I have referred above to the persistence of the response after experimental or pathological interference with the anatomical substrata of nervous activity, but want to point now to an even more impressive fact, namely, the great variability in the degree of precision of the anatomical networks in the course of development. The fact is that we frequently suspect a given neuronal precision setup as being relevant for a particular neural function, but often find that in an earlier stage of development this function will be performed in essentially the same way without that particular structural precision scheme having even developed as yet. In general, many a condition which we would think essential from the study of the developed nervous system loses pertinence when studies of earlier stages show that things work very much the same even in its absence. This must be emphasized particularly in connection with the present discussion of the relation between input and output of the nervous system. It is a fact that most of the basic motor patterns of behavior are developed within the nervous system by virtue of the laws of its own embryonic differentiation without the aid of, and prior to the appearance of, a sensory input from the outside world. The basic configuration of the motor patterns, therefore, cannot possibly be a direct product of the patterns of the sensory input. A study of the development of the nervous system and of behavior forces us to consider the output of the nervous system and its patterns as primarily preformed within the nervous system and ready for use, requiring the sensory input for release, facilitation, and modification, but not for its primary shaping.

This brings us to the fundamental alternative, to which I think Dr. Gerard has likewise referred, of whether the central nervous system is merely a clearing house for input-to-output messages, or whether it generates activities of its own and has patterns of activities of its own, the elements of which are not pieced together by, and reflections of, the sensory input. Dr. Köhler has likewise touched on this fundamental difference in the interpretation of the realities of the nervous system. No theory of the nervous system can claim to represent the facts if it ignores the central autonomy of the basic patterns of motor performance. This autonomy impresses us not only in the studies of the development, but also in studies on reconstitution after injury of the nervous system, which touches on a question Dr. von Neumann has raised with regard to learning. This is the question of whether learning implies a complete reorganization of the nervous network with a resetting of relations among individual neurons, or the acquisition of a new performance, which will merely supersede, rather than replace, the older performance. This question can be crucially studied by disarranging the peripheral motor apparatus by crossing tendons or nerves, and thus rendering the original impulse patterns inadequate for the performance of a given act. Experiments by my former student, Sperry, have shown that rats cannot relearn their motor coordination to meet such new situations. Studies we have made on patients with transplanted tendons after partial infantile paralysis show that they can learn to use the transplanted muscle in its new function, but precise electro-myographic records show that the muscle is apt to lapse back into its innate phase of activity, thus proving that the learning act does not dissolve the original patterns of motor organization. Evidence of this kind demonstrates clearly that the act of learning does not consist of merely a recombination of individual neuronal elements. On the basis of all existing evidence, the nervous system must not be conceived of as a network of monotonic elements, but as a hierarchical system in which groups of neuronal complexes of different kinds are acting as units, the properties of which determine the configuration of the output pattern. Some of these higher units are rigidly fixed in their functions, others are modifiable by experience. I fail to see this hierarchical principle duly reflected in the theory of a monotonic network of units such as has been discussed in this session.

. . . .

DR. WEISS:[*] Let me start the present discussion by stressing the great change in our thinking which the facts we have been hearing mark over the state of affairs twenty or twenty-five years ago, when the hard, fast, and precise one-, two-, or three-neuron arcs were viewed as the keys to the understanding of nervous activity. In the meantime we have come to recognize the tremendous complication of the system. This development ought to caution us against trying again to draw a final picture with the material available to our present fragmentary state of knowledge. We ought to expect that in another twenty-five years additional essential parameters will have been discovered. We have been impressed here with the tremendous complication of the nervous network as far as the shape, distribution, and ramification of the individual elements is concerned. To this intricacy of spatial patterns, I am sure, will have to be added, as another parameter determining nervous activity, specific constitutional differences of biochemical kind among the elements, as well as specific states of activity of larger collective units based on those chemical differences. If I may briefly elaborate this concept, we have been treating the nervous system as essentially composed of units of a single kind. Dr. Lorente de Nó has emphasized the existence of a great variety of what he calls "types" of which only three or four have thus far been studied, while thousands of others are still to be explored.

---

[*] The discussion to follow (through page 82) arose out of Dr. Lorente de Nó's paper. A good deal of that discussion was so specific to the paper as to be meaningless without it and so is omitted; the remainder is included here. EDITOR.

But what is really meant by a cell type? Does the term merely signify a characteristic shape of the cell body, distribution of the processes, and geometric configuration of the arborizations? Or does the term mean to imply the existence of different cell species with distinctive chemical properties? Is the protoplasm of all nerve cells the same, merely cast into different forms and arrangements? Or do these different types of nerve cells have distinctive chemical characteristics that are instrumental in the operation of the nervous machine? Evidence is increasing that we shall have to adopt the latter view. The variety discernible under the microscope is merely one of the relevant differentials among nerve cells. If we had only microscopic pictures of different glandular cells, we might consider them to be of one kind, but we know that their products and their production machineries differ fundamentally in chemical regards in spite of their similar appearances. Likewise the microscopic resemblance of all nerve cells is no evidence of their similarity chemically. There are two problems to be faced. First, are there significant chemical differences among neurons? Second, if so, what is their role in the operation of the nervous system? As for the existence of chemical differences, the evidence seems clear. Pharmacology has demonstrated that different drugs have a selective affinity for different types of nerve cells. Such different response to chemicals can only be based on a fundamental difference in the chemical constitution of the responding neurons. Similarly, when we classify neurons as cholinergic or adrenergic, that is, as capable of producing different types of substances, we admit that the fundamental constitution of their protoplasms must be different. There is evidence that different types of nerve fibers, such as sensory and motor fibers, and perhaps even different subclasses of fibers within each category, follow different routes in their primary outgrowth during development. This again expresses constitutional chemical differences between those types, endowing them with selective chemical affinities by which they can follow different tracts. Again in establishing peripheral connections with effector or receptor organs, and similarly perhaps between different intracentral junctions, selective discriminatory faculties come into play which can only be conceived of in terms of specific chemical sensitivities based on the chemical organization of the respective units. The existence of chemical differentials between neurons has thus been established beyond doubt. The question then arises whether they may be dismissed as simply instruments in the structural development of the nervous system, which leave their residues in the adult body but are irrelevant as far as the operation of the nervous system is concerned, or whether such chemical differentials are significant factors in nervous activity. That is, do we have to add chemical parameters as determiners of nervous activity to the known parameters of the conduction process, such as time sequence, frequencies, thresholds, synchronism, and other space-time characteristics based on the electric activity of a characteristically distributed network? I submit that we must answer in the affirmative, that we must recognize qualitative differences among neurons as decisive in the making and breaking of functional connections, and that chemical conformance or nonconformance between elements may decide whether a synaptic junction will be passable or impassable.

This brings into view a whole new field of possibilities, which it seems can derive substantial support from general biological experience. It may be appropriate to point out here this more general biological background. As I have outlined in the past several years, many relations between contiguous cells are explicable in terms of the conformance or non-conformance between the configurations of the molecular surface populations occupying the contact surfaces. That is, the behavior of two contiguous cells will differ fundamentally, depending on whether or not molecular key species on either side of the contact surface interlock by virtue of their steric relations. We may visualize these relations in the old key-lock picture of Ehrlich in the modern version given to it in Professor Pauling's theories or in any similar fashion. Such relations are the basis of enzyme reactions, of serological reactions, presumably of growth and reproduction, and as I have tried to show, of intercellular relations. If specific intermolecular bonds along the surface of cells are the underlying mechanism of cellular affinity in general, then it would be quite plausible to assume that the making and breaking of connections between neurons might likewise depend on the conformance of the molecular patterns along their surfaces. That is, given a stationary microscopic network of connections, the impulse could pass only from one element to another if the molecular populations along the barrier were of matched character. Since, as I have indicated in another place, the molecular border population of the cell will vary, depending on the physiological state of that cell, a cell with a given chemical constitution can assume a considerable variety of surface states and accordingly play different parts in intercellular and supercellular activities. It is possible that the proper setting of these border conditions among contiguous elements forms a process quite distinct from and preparatory to the conduction process, and if this is true, then you realize that we have hitherto omitted a very significant coordinating principle from our nervous theories. To the parameters of space and time may have to be added those of chemical conformance and non-conformance. In this connection I should like to call your attention to the fact that in this morning's discussion Dr. McCulloch has illustrated his theory solely by examples taken from those sensory fields which have spatial organization, that is, the optical, tactile, and acoustic fields. In these cases, it is relatively easy to make a case for the patterning of the response by switch-work systems.

However, when we turn to non-spatial qualities of sensation, such as color, taste, or smell, the resolution of these qualities into spatial distributions certainly presents difficulties that cannot be glossed over. Let us consider for instance the experiments of Curt Richter in which rats proved capable of selecting among a variety of foods the one most appropriate to an artificially produced deficiency state of their bodies. Or take the ability of a parasite to recognize very specifically its prospective host by certain chemical cues of the latter. It seems to me impossible to account for these facts otherwise than by conceding to the chemical specificity of the neurons a decisive role in the operation of the system. If in the future we will focus attention on the chemical differentiations among neurons as the presumable mechanism of establishing and breaking intracentral functional relations, we shall undoubtedly find more supporting evidence for this concept. Of course, the microscope will not help us in this task except in so far as a specific differential stain is frequently a safe indicator of biochemical diversity among cells.

DR. BROSIN: This striking development certainly deserves more discussion. Perhaps we can persuade Professor Pauling to comment on it later in the week.

DR. GERARD: Since I have been insisting for years that there are chemical differences between neurons in the different systems, I am obviously, in general, in complete sympathy with the approach which Dr. Weiss has just urged, and I think it might be stimulating to apply it to the question of memory. If that should depend on the passage of impulses across synapses, thereby leaving them altered, might not the alteration, in fact, be a kind of molecular reorganization of the surfaces at the junction, of the sort suggested? I am not inclined to be too optimistic about this extra freedom helping us greatly at the present time, at the level of analysis which is possible with the present procedures—even the chemical ones. Unless one could progress by logic, there is the problem of getting antigens out; and it would be pretty terrific if evidence were obtained that neurons can interact only if they are of the appropriate chemical specificity to match with each other. I think this can perhaps be dismissed at the present time on three grounds. One, although in normal function many neurons are functionally inaccessible to each other, with appropriate drugs they do become accessible to each other—as in strychnine poisoning. Two, even in the normal reflex chains, cholinergic preganglionic fibers act on adrenergic postganglionic fibers. And, third, even within a single neuron itself, there is unquestionable chemical specification, the metabolic reactions of nerve cells being different from those of the nerve fibers which come from the nerve cells. One prefers glucose as fuel and the other glycogen. Cortex is inhibited differentially more by iodo-acetic acid and medulla by malonate. And even in degenerative diseases, the absence of one of the vitamin B components leads to degeneration in the cell bodies of a sensory neuron group, the fiber degenerating only secondarily; and absence of another of the B components leads to degeneration of the fibers, with the cell bodies only secondarily showing disturbance. Therefore, I doubt if we can make too much of chemical specification as a basis for function at the present time, however valuable it may be in interpreting time and space sequences in the development of brain, nerve, muscle, and the like.

DR. KLÜVER: It is of interest that some of the older neuroanatomists, more than thirty years ago, insisted on the importance of chemical differences in the nervous system. In fact, they provided data in support of a chemical topography by systematically studying the absence or presence of various substances in different regions of the nervous system or in different types of cells and fibers. Although the methods used may seem crude in the days of modern cytochemistry there is no doubt that the importance of chemical differences for the study of normal and pathological functions was recognized long ago. When I once discussed these matters with my friend, Dr. Polyak, I ventured the opinion that each nucleus in the brain stem differs chemically from every other nucleus. He not only agreed, but insisted that each neuron differs chemically from every other neuron. When I asked him for his reasons he replied, "All neurons have different *shapes.*"

DR. WEISS: My reference to chemical differences among nerve cells has not been altogether understood. The gross detectable differences which I mentioned and to which may be added those of preferences for nutrient substrates and other metabolic differences, I have used only as crude indicators of the existence of differentials; but the relevant differences which I think are instrumental in the operation of the nervous system are of much subtler character. We cannot even be sure whether these differences concern the chemical composition of the cell or merely the relative distribution of the chemical key compounds present in the cells, since what determines the behavior of a cell under given conditions is only the particular segment of the molecular population that happens to occupy the surface and thereby controls what is going into and out from the cell as well as what impulse relations the cell will establish with its neighbors. Such subtle differences of organization will hardly ever be detected by grinding the cell up and making chemical bulk determinations. The main purpose of my remark was to call attention to the presence of such subtle chemical relations as instrumentalities in biological relations in general and to call for more intensified study in the case of the nervous system. I may add another biological comment, returning to Dr. Lorente de Nó's remark that his identification of cell types referred to characters of shape and spatial organization. The study of developmental mechanics has shown that the shape of a cell is only an expression of responses of the cell to its environment in the course of its development. Thus if two cells in an otherwise identical environment assume different shapes, this can generally be considered an expression of underlying differences in the response mechanisms of these cells, or in other words, of differentials in their physiochemical make-up. This is further evidence of the constitutional diversity of the cellular elements in the nervous system.

DR. BROSIN: I have asked Dr. Wiersma of the Biology Department here to tell us of some of his work related to the problem under discussion. Dr. Wiersma.

DR. WIERSMA: I'm glad to have this opportunity to bring before you some work I have done with the synapses of the crayfish central nervous system and at the same time to put some questions to Dr. Lorente de Nó that occur to me concerning the influence of the structure of synapses on transmission. The central nervous system of the crayfish with which I have been working is a rather fortunate contribution of nature, in that it offers, for one thing, a postganglionic fiber which can be stimulated by different preganglionic elements, three of which can be isolated and stimulated as single fibers. These elements are the giant fibers, which run through the whole length of the central nervous system. On stimulation with a single shock, the resulting impulse in any of the giant fibers causes a discharge in a motor root of each of the abdominal ganglia, and the animal flaps its tail. That synaptic transmission is involved can be shown by the fact that stimulation of the motor root does not give rise to activity of the giant fibers. The synapses between the giant fibers and the fiber in the root which discharges have been described by Johnson (1924). If one makes a cross-section of the cord, one will find that the motor root fiber, which itself is of sizable diameter in the root, arises from a ganglion cell on the opposite side. It travels straight up and makes first contact with the heterolateral medial, then crosses over to the homolateral medial, next lays itself against the homolateral lateral giant fiber, and then enters the root. The places of contact are most likely the places where transmission occurs. We find that indeed, by leading off from the root, the root potential follows closest after the impulse in the homolateral lateral fiber, somewhat later after the homolateral medial impulse arrives, and the greatest delay occurs when the heterolateral medial is the activating fiber. Stimulation of the heterolateral lateral fiber always results in excitation of the homolateral lateral, since these two fibers are connected in each ganglion by a synapse, which has some special properties of its own which time does not permit me to describe.

Now, it is possible to bring the preparation into a state in which a single impulse in any of the giant fibers will no longer result in a root potential. Under these circumstances, we will find that if we put two impulses in any one synapse, a root potential will result for time intervals up to about 15 milliseconds. This is, of course, a fairly long time for synaptic summation. However, there is a stranger phenomenon: We can, when the preparation is in this state, combine the stimulation of any two giant fibers and obtain within certain time limits, root potentials. Another remarkable fact here is that simultaneous stimulation of the postganglionic fiber at two synapses does not in general result in a root potential, but that the two preganglionic impulses have to arrive with at least a certain interval. The period during which no root potential is obtained I have called the inert period. If we combine two neighboring synapses, this period has a length of about 2 milliseconds, whereas if another, inactive synapse intervenes, its duration is about twice as long. At the same time the period during which summation can be obtained is shortened with increased distance between the synapses. In other words we may conclude that a process must spread with a certain speed and diminishing intensity from one synapse to the other.

There is pretty good evidence that excitation indeed takes place at the site of the secondly stimulated synapse and not somewhere between the two synapses. In the case where a single impulse causes a root potential, there is, as mentioned, a difference between the delays, when different giant fibers are stimulated. Similar differences are present when summation is necessary, and the delays obtained in this way correspond best with those of the second fiber stimulated. There must thus be a conduction of some process which causes increased excitability. The nature of this process is uncertain: chemical, electrical, or a combination. From the duration of the inert periods and the distance between the synapses, it is possible roughly to calculate the speed of spread, and this comes out as about one fiftieth of the normal conduction speed of the action potential of the root fiber. There is still a fourth synapse, of which less is known, by which the root fiber may be stimulated. This one can be stimulated by exciting the first root on the same side as the third root, from which the lead is taken. Since the whole first root is stimulated, there may be more than one synaptic connection involved in this case. Nevertheless, in combination with giant fiber stimulation, first root stimulation gives results which fit well into the picture. Thus the inert period is longest when the distance is greatest, which is the case when lateral giant fiber and first root stimulation are combined.

The synapses described previously are by no means the only interesting ones which the crayfish has to offer. There are also the synapses in the lateral giant fibers. These are reminiscent of similar structures in the giant fibers of the earthworm, as worked out among others by Dr. Bullock. These macrosynapses consist of a joining of two nerve elements of the same diameter by a membrane. I have tested this membrane in the lateral fibers mechanically. By pinching the fiber below the membrane the end will swell up quite considerably, but no fluid will go into the anterior fiber. Also, on pulling, the two fibers do not come apart. There is, for all practical purposes, a complete anatomical division between the two nervous elements. Nevertheless conduction takes place in both directions with equal facility. My opinion is that this may be due to the wide contact area which this synapse forms. In the above discussed synapses, the postganglionic fiber is much smaller than the preganglionic and hence a large part of the circumference of the postganglionic fiber is involved in the synapse, but only a limited area of the giant fiber. The latter factor would make it impossible for the action potential of the root fiber to stimulate the giant fiber, and hence one way conduction. I would like very much to know from Dr. Lorente de Nó, whether or not he would make such purely anatomical differences responsible for differences between other synapses.

DR. MC CULLOCH: May I ask one question? In this buck-jointed axon, did you say there is or is not any synaptic delay?

DR. WIERSMA: I do know that if there is a synaptic delay it is less than one tenth of a millisecond. One cannot say there is none, because the measurements are not more accurate than this.

DR. LORENTE DE NÓ: Is the mechanism by which you change the state of the ganglia cells rapidity of stimulation?

DR. WIERSMA: Yes, but aging will have a similar effect. Once brought into this state they remain in it for a time. The delay is also not constant in these preparations. The longer you stimulate, for instance with an intermediate frequency of 20 per second, the more the synaptic delay will increase, until the root action potential drops out.

DR. BROSIN: Thank you, Dr. Wiersma. Dr. Lashley?

DR. LASHLEY: I should like to point out the wide ramifications of the work of Dr. Lorente de Nó, work which has been revolutionary in many fields of study of the nervous system and of psychology. In the first decades of this century, the view was widely held (at least among those of us who are somewhat naïve in neurophysiology) that the conduction in the nervous system is always downstream. As a result of that view, in large part, came the development of behaviorism; the denial of imagery (memory images) because there seemed to be no mechanism for central reinstatement of activity, and the development of motor theories of thinking by Watson and others. There seemed to be no mechanism for continuity of thought processes except a chain of circular reflexes involving muscular contraction and the resultant stimulation. Many theories of learning at that time were also very strongly influenced by this conception. The dominance of the belief that the conditioned reflex is the primary form or prototype of the mechanisms of learning derives in large measure from this concept. Dr. Lorente de Nó, in his demonstration of reverberatory circuits, provides a mechanism for central maintenance of activity and so throws an entirely new light upon many of these problems. His studies are now leading to a rather widespread revolution in psychological as well as in neurological concepts.

. . . .

DR. BROSIN: With the thought that we would continue the discussion of the previous speakers in as unified a form as possible this morning, we will pay Dr. McCulloch the most sincere of compliments by working hard at re-examining his propositions. Therefore, continuing what we began earlier, I will call on Dr. Liddell to tell us about the difficulties in methodology of animal behavior as it relates to the neurophysiologist.

DR. LIDDELL: Dr. McCulloch's paper, as the discussion of yesterday demonstrated, was admirably provocative and, in my own case, led to disquieting nocturnal ruminations concerning psychology in its present relation to neurophysiology. May I, therefore, give a sheep's-eye view of the matters under discussion; that is to say, from a strictly pastoral point of view.

In the first place, most of the experimentally observed facts of neural function and the structural details upon which Dr. McCulloch's theoretical formulation is based are derived from mammals simpler than man. Why, then, would it not prove more rewarding to attempt to fit facts derived from systematic experimental observations of the behavior of the simpler mammals—sheep, goat, cat, dog—to the theory rather than select instances of human behavior to support the theory? This can be done and should be done.

I propose Pavlov's method of the conditioned reflex as almost ideal for the purpose. Conditioned reflex study has for too long been misunderstood and for a simple reason. Pavlov's method has yielded a mass of verifiable facts in search of a theory. Dr. McCulloch's admirable theory is in search of facts. Is it not possible that, first, Pavlov and, now, McCulloch suffer from a painful phantom limb? In Pavlov's case the phantom limb was an oversimplified imaginary nervous system which could not support the weight of facts. Perhaps McCulloch's painful phantom is the colorless ghost of real behavior. Let us see how we may relieve McCulloch's suffering.

Gasser has said that the electrophysiologist can answer the question "when?" but not "what?" Here is precisely the advantage of Pavlov's method. The conditioned animal can be trained quite exactly to tell *when* by salivation, leg flexion, cardiac acceleration, or other physiological indicators. Moreover, we can fairly exactly explore the operational limits of its neural computing machine through discovering which forms of trained anticipation lead to breakdown or experimental neurosis. We can find its most vulnerable spots.

The sheep, goat, or dog, trained to self-imposed restraint, through the use of a comfortable restraining harness, learns to expect electric shock or food, or acid in the mouth, as Pavlov found, at some signals but not at others. Some dogs, but not all, when thoroughly conditioned to a series of positive and negative signals, may develop experimental neurosis when the signals are suddenly reversed, where, now, all food signals mean "no food," and "no food" signals are all followed by food. We have verified this neurosis-producing procedure in the pig. However, a slight change in Pavlov's method of reversing signals will render it innocuous. If a sheep is trained for several months to expect mild shock on the foreleg following a buzzer sounding for 10 seconds, and no shock following the ringing of a bell for 10 seconds, these signals may be reversed with impunity twice within a single two-hour period by proceeding as follows. The buzzer sounds; the sheep alerts; but just before it is able to raise the foot from the platform, the buzzer ceases and no shock follows. The bell sounds for a few seconds, and shock is given. In very few repetitions of this cut-off procedure, the sheep has correctly adjusted to the reversed signals—flexing the leg at the bell and remaining quiet at the buzzer.

Another simple method of testing the sheep's and the goat's neural computing machine is as follows. A telegraph sounder clicks once a second for 10 seconds and a shock is applied to the foreleg. After 2 minutes, the telegraph sounder clicks again for 10 seconds, followed by shock. Each day, twenty signals of 10 seconds' duration are followed by shocks, with 2-minute intervals between signals. Neurosis supervenes after about one thousand signals at twenty per day, and persists with no further training for as long as three years. It is characterized by muscular rigidity and slow heart. Where the separation of 10-second signals is by constant 5-, 6-, or 7-minute intervals, neurosis again supervenes; but in sheep and goats, it is a strikingly different neurotic pattern, characterized by diffuse motor activity and rapid, irregular heart. In both types of neurosis, the animal's behavior in the pasture and in the barn is radically changed, with a loss of gregariousness, and with restlessness during the night. If the 10-second signals followed by shock are separated by 1-minute intervals, the animal will submit to as many as forty signal shock combinations per day with no signs of perturbation. Furthermore, shocks spaced at regular intervals of 1 to 7 minutes leave the animal quite undisturbed, although it conditions to the true interval as such, and flexes the limb just before the shock is given.

Work along these lines is technically simple but time consuming. It is, nevertheless, worth the investment of time if it integrates, as I think it does, with the technically more formidable tasks undertaken by Dr. McCulloch in furthering understanding of cerebral mechanisms in behavior.

DR. KLÜVER: Dr. McCulloch told us yesterday morning that he wanted to stay away from the totality of behavior. I am very glad indeed that he didn't make good his threat. As far as I can see, he discussed practically every aspect of behavior, since he considered reflexes, audition, pattern vision, learning, memory, thoughts, ideas, goals, motivation, will, and consciousness.

First, I should like to have some additional information on a few points. You mentioned that stimulation of the primary visual cortex in man elicits the impression of a blurred circle of light whereas stimulation of a spot in the secondary visual cortex leads to the impression of a form. There are reports in the literature indicating that the nature of the photopsiae varies with the type of electrical stimulation employed. It has been reported, for instance, that the photopsiae consist mostly of glowing and colored "roundish forms, disks, or rings" when faradic current is employed, and of stars and ragged forms, such as "pointed sparks," when galvanic current is used. I am wondering whether you care to specify the experimental conditions under which the results you mentioned were obtained. In analyzing the mechanisms of hallucinations, I was interested in the question whether unformed hallucinations ever occur, that is, hallucinations of mere luminosity. It seems that reports on hallucinations never refer to the appearance of visual "dust" or to an undifferentiated luminosity. I am wondering whether you know of any reports referring to unformed hallucinations.

DR. JEFFRESS: Dr. McCulloch also mentioned that the form aroused by stimulating area 18 had a location in space. Was the location relative to the head or to the outside world? You said that it was not relative to the eyes.

DR. MC CULLOCH: It is not located in the visual field, because the patient can turn his eyes toward it. It does not move with the eyes, but stays there so that he can direct his eyes toward it. This is quite different from what is seen when area 17 is stimulated or in entoptic phenomena.

DR. KLÜVER: In connection with your analysis of the reflex turning of the eyes, it seems pertinent to recall Graham Brown's observations on the reflex orientation of the optical axes. He observed, for instance, that the optical axes remain fixed in space if the head of a moderately narcotized monkey is passively moved to one side. This reflex disappears upon removing the superior colliculi or upon increasing the depth of narcosis. It is found to be present in monkeys which are decerebrated at the cephalic boundary of the superior colliculi. Graham Brown was chiefly concerned with determining the influence of cortical activity on this reflex orientation of the optical axes. His work culminated in the suggestion that eye movements obtained from the occipital lobe are equivalent to those which, in normal life, center the visual image of an object fixed in space by bringing it from the peripheral retina to the central field of vision, while movements obtained from the superior frontal convolution correspond to those which, in normal life, keep the optical axes fixed on an object that is moving in space. Graham Brown also suggested that movements obtained from the middle frontal convolution are equivalent to those in normal life where the eyes move at command. I am wondering whether you have any electrophysiological data supporting Graham Brown's idea of such entirely different mechanisms in eye movements.

As regards the general problem of relations between cortical mechanisms and behavior, I am glad, as I said before, that Dr. McCulloch has touched upon so many diverse aspects of behavior. He might be accused, and he has been accused, of talking about behaviors that are very tenuous forms of behavior or even forms of behavior that do not actually occur. Nevertheless, I am hopeful that his analysis may lead to a profitable search for "phantom limbs" and ultimately to full-fledged behavior realities. It is possible that certain forms and consistencies of behavior will be discovered that would escape detection without his kind of analysis.

Finally, I should like to return to the subject of "ideas." It is not clear to me whether you are content with proposing certain cortical mechanisms that are operative while I am having an idea or whether you really want to write a neurophysiology of logic. You recall that Brickner, some years ago, found that electrical stimulation of a cortical area on the mesial surface of the left hemisphere, at about the junction of Brodmann's areas 6 and 32, resulted in perseveration of speech. When his patient was requested to recite the alphabet she repeated again and again the letter she happened to say at the moment this area was stimulated: A, B, C, D, E, E, E, E, E, F, G, H, I, J, J, J, J, K, L, etc. The repetition continued as long as this area was stimulated and ceased immediately on terminating the stimulation. Perhaps we have here a beautiful demonstration of the workings of a reverberating circuit. A psychologist may have no difficulty in relating circuits of such kind to perseverating ideas. The general point I wish to emphasize is that having an idea, or having even an idea that is perseverating, is different from an idea. Ideas, logical structures, and meanings are in and of a world that is different from the world of physiological and psychological events that occur in the process of having ideas or recognizing and enunciating propositions and meanings, even if such physiological events should represent events in reverberating circuits. I am, therefore, still curious to know whether you want to contribute to a neurophysiology of logic or merely to a neurophysiology that is involved while I am talking about logic.

DR. LINDSLEY: I believe I understood Dr. McCulloch to say, at one point, that he interpreted the alpha rhythm as a possible fluctuation in the excitatory level of the cortex, particularly with reference to the speed of transposition, say from one chord to another in an auditory field or from one pattern to another in the visual field.

DR. MC CULLOCH: That is correct.

DR. LINDSLEY: It is a little difficult for me to see how this could be the case inasmuch as visual stimulation will completely block the alpha rhythm for a period, even after the stimulation has stopped.

Another thing: An experiment not yet reported which was done by Dr. J. R. Smith and myself just before the war (it was interrupted and not continued since—I hope to get back to it) had to do with visual stimulation in relation to modifications of the alpha rhythm. Briefly, it was this. A small spot of light which could be controlled in intensity and in duration, was thrown on the periphery of the retina, and we investigated the effects of its intensity and duration on the blocking of the alpha rhythm. When we got down near to threshold with our stimulation and beyond the subjective threshold, we still got effective blocking of the alpha rhythm. We were recording with our electrodes over areas 18 and 19, so probably very little of area 17 was involved in the case of electrodes on the human head. We were having our subject report after each flash came, and they came at irregular intervals for which he was not warned. When we got to threshold the subject's report would cease. Nevertheless, for a certain region below the subjective threshold, the blocking would still occur. Now, what I would like to have Dr. McCulloch explain, in terms of the McCulloch-Pitts model, is this separation, presumably, of the awareness of the stimulus on the part of the subject and the physiologi-

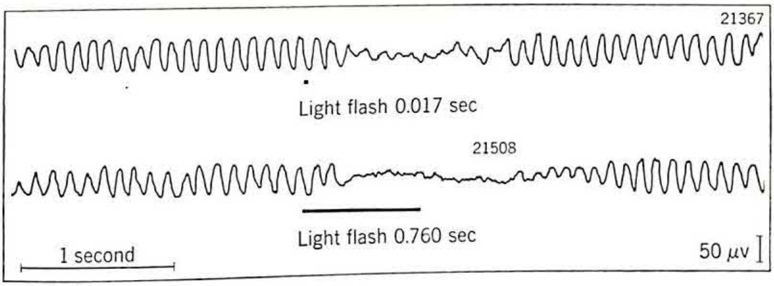


FIGURE 1. Effect of a light flash upon alpha rhythm.

cal response that we get. Figure 1 illustrates the kind of blocking that one gets.

The illustration does not show what I have just been speaking of —that is, the separation of the awareness or non-awareness of the stimulus on the part of the subject. It simply shows the kind of response we get from a flash of light, in this case, one of which the subject is definitely aware. The duration of the stimulus patch represents the duration of the flash of light. In this illustration, both flashes of light were of the same intensity but of different duration. The point I want to call attention to, however, is that we get this kind of blocking whether the subject is or is not aware of the light, in this region just below the subjective threshold.

DR. MC CULLOCH: I wonder if Dr. Halstead would in this connection be good enough to mention his work on the driving of alpha by light. There are probably many people here who don't know the story.

DR. HALSTEAD: If I may postpone it for 30 minutes or so I'll get a slide illustrating the effect.

DR. KLÜVER: Is the duration of the blocking dependent upon the intensity characteristics of the stimulus?

DR. LINDSLEY: It is related to it, but it is related more, perhaps, to the duration of the seen stimulus. In the illustration with the brief flash, after about two-fifths of a second, the blocking begins, and you can see that it persists. Many times you will get really a summation or an after-effect following the cessation of the stimulus. In the second part where the stimulus persists for a longer period of time, you will again see that there is a period following the cessation of the stimulus during which the alpha waves remain blocked and only gradually return, and there you get some of the increased amplitude effect.

DR. KLÜVER: Does the fact that the duration of the blocking is dependent upon the duration or the intensity of the stimulus imply that the awareness or the non-awareness of the stimulus plays no role in influencing the duration of the blocking?

DR. LINDSLEY: I think it is related to both time and intensity.

DR. KLÜVER: Suppose you are able to relate the duration of the blocking to the product of time and intensity. Does this imply that the duration of the blocking is not influenced by awareness or non-awareness on the part of the subject?

DR. LINDSLEY: I don't believe that we have the situation well enough defined to say just what the duration of blocking means as yet. We have in mind further investigation of the problem.

DR. MC CULLOCH: Jasper has a good deal of evidence with anxious patients to show that you get disproportionately long disappearances of alpha on very dim illumination even if the patient doesn't see it, and that the duration parallels the severity of the sympathetic overactivity of the patient generally.

DR. WOODBURY: Just to complicate the issue a little further, I would like to mention our experience on the disappearance of the alpha rhythm, depending on whether or not you are getting pattern vision. With electrodes over the occipital region and a towel over the eyes, and the eyes open, we get alpha rhythm with the eyes blank. When we request the subject to focus his eyes on the towel pattern, the alpha rhythm disappears.

DR. BROSIN: Dr. Weiss, do you wish an opportunity to enlarge on this topic?

DR. WEISS: May I mention a few features manifested by nervous activity which any valid theory of the nervous system must duly take into account?

First, the nervous system uses in its operation certain distinct specificities: a specific receptor type and, on the effector side, a specific muscle. Many of you have seen my film on myotypic function in supernumerary limbs. I have demonstrated the same specificity in the proprioceptive system and for the corneal reflex, and Sperry has extended the evidence to the optic field. The existence of such specificities through which discrete peripheral organs and their corresponding central activities are related is an incontrovertible fact. The interpretation, however, is a matter of speculation. Whether we look for spatial order, chronological order, or chemical order as a basis of these specificities, we must always bear in mind that they are distinct, discrete, and show no intergradations. They could be called qualitative differences. Personally I still like to view them as resonance phenomena between complex molecular elements of matching configuration or tuning.

A second condition that any theory of the nervous system must satisfy is that it must be able to explain perception not only in spatially extended fields, such as tactile, acoustic, and optical space—those used by Dr. McCulloch—but must equally well explain recognition of color and chemical character. I submit that this can be done if we concede to the elements of the nervous system the power to recognize chemical constitution by virtue of their own chemical characteristics.

A third condition is that the theory must give equal regard to the role of the molecular processes, the microscopic cellular organizations, and the field properties of the nervous system, all of which seem to be demonstrably involved. How can we reconcile our view that neuronal relations on the molecular level are a coordinating principle with the recognized fact that each act involves vast numbers of neurons whose collective activity behaves in the manner of continuous patterned fields? Perhaps the answer lies in a dualistic concept of nervous activity. We may assume one activity of a conditioning type that sets the stage for a given reaction by determining molecular interneuronal interdigitation, thus linking definite sections of the neuronal network into closed systems, within which, then, a second activity with field character, perhaps merely of electrical order, would operate. Thus by molecular relations, functional subdivisions would be carved out from the total pool of the nervous system, and each such subdivision would then operate field-fashion. Groups of such higher units might then become linked in their activities through correspondences in their respective field activities, and in this fashion we could arrive at a physical counterpart of what we have called the hierarchical principle of central activities. The field character of the activities in neuron pools may force us to extend our current concept of the nervous system in another direction. We may find that the field properties cannot be adequately accounted for by considering only the neuronal elements, that is, by equating the nervous system with the sum total of its neurons. It may become necessary to consider the possible role of the matrix in which the neurons are embedded and learn more of its properties. The environment of the neurons is by no means a physiochemically or anatomically simple system, but is a tissue of complex structural and physiochemical organization. The structural and biological properties of this matrix will of necessity affect the processes in the neuron circuits which are embedded in it and perhaps in counterplay with it. This proposition, perhaps first envisaged by Nissl, certainly deserves further exploration.

A fourth demand on any valid theory of the nervous system is that it must not only apply to the developed operative nervous system, but must explain why the nervous system does not change in its essential functions during growth, although its anatomical substratum is in constant expansion. One must bear in mind that as the minute embryo or fetus or larva grows to adult proportions, the nervous system undergoes enormous alterations in mass, dimensions, interneuronal relations, in the numbers of cells and their sizes. The crucial factors are that the patterns of performance remain relatively stable in spite of these variations of the underlying substrata. New performances may be added, and the precision and speed of older performances may be increased as development goes on, but the basic patterns of coordination remain remarkably constant in spite of the progressive distortion of the underlying anatomical substratum as a result of anatomical growth. This fact brings into sharp relief a question which we have discussed earlier, namely, just how much precision does the nervous system really apply in its performances? If I understand Dr. McCulloch correctly, he tries in his concept to replace mechanical and detailed precision by statistical normalization. I think this is a very fundamental consideration, not only in psychology, but also in biology in general, particularly as regards the processes of development. I wholeheartedly concur in this attitude and hope that it will be more generally adopted. We focus too much attention on precision arrangements among elements, such as a preordained length of a collateral, a rigidly fixed number of collaterals and end feet, a precise diameter of a fiber, and so forth, instead of basing the regularity of a performance on the statistical properties of the collective of units exhibiting it. The regularities observed must be based on the normalization of the statistical distribution of properties in the collective rather than on the minutely precise arrangement of details. This leads us to recognize a certain degree of indeterminacy in biological microevents. This has been true of development in general, and now it seems to be equally true of the operation of the nervous system.

A fifth, and the last condition I want to mention, is one already outlined earlier, namely, that the patterns of motor coordination are not replicas, as it were, of the sensory input patterns. The character and the structure of the motor output is determined by the intrinsic properties of the organized response system and not by the set of stimuli that are fed into it from the environment or the surrounding body. With Dr. Köhler I, too, take exception to calling the light perception, obtained upon pressing on the eyeball, a wrong or false impression in contradistinction to one released by radiant energy. I think it is just as much the real thing as a visual excitation. It is a manifestation of that constitutional ability of the central optical apparatus to generate an activity sensed as light, whether stimulated "adequately" or not. However, whatever the situation in the field of receptors, there is positive evidence that in the motor field, at least, the structural patterns of coordinated response are shaped and differentiated prior to, and without the aid of, a sensory input that could have impressed its structure upon them.

DR. PAULING: This problem of cerebral structure and mechanism may well be related to my field of interest—molecular structure and molecular mechanism—indeed, it must be, as Dr. Weiss has pointed out. I would like to know the order of magnitude of the dimensions of the structures of primary importance in cerebral mechanisms. I suppose (I don't really know anything in this field) that memories—as cerebral processes—involve at first electrical patterns, electrical structure, and that then, after the passage of a suitable period of time, these electrical patterns may be transmuted into material patterns—material structure. And so we ask: How is it possible for this change from electrical structure to material structure to occur, and what is the order of magnitude in size of these material structures? I am not much interested in large structures—structures a hundred angstroms or a thousand angstroms in linear dimensions—but, at least at present, in smaller ones. We can divide things up into different categories of size. First we may consider structures which are, say, 2 to 10 angstroms in dimension—atoms and little groups of atoms, such as the carboxyl group, or an amino-acid residue, that determine mainly the chemical properties of substances. Then we have the next category from about 10 angstroms to, say, 40 angstroms in size. These structures in the main determine, I think, the specific biological and physiological properties of substances. I think that biological specificity is determined almost completely by the size and shape of the outsides of molecules and especially by the phenomenon of complementariness in structure—a sort of lock and key effect. Two large molecules may show a specific relation to one another because of a complementariness in structure. Immuno-chemical experiments have shown that the complementariness must be good to within an angstrom or so, the areas involved being of the order of magnitude of 10 to 20 angstroms square.

Then we have the next category of dimensions, from 40 to 500 or 1000 angstroms, and finally we reach the category of large structures—microscopic structures. This category of structures is extremely important in biology generally; but is it important in a significant way in cerebral processes? I doubt that it is. I think that it is related to cerebral processes, but that it is not important in the sense that cerebral processes involve a change in structure in this size region.

I believe that the specificity of antigens, enzymes, and genes depends on structure in the 10 to 40 angstrom region, that mutation involves a change in structure in this region, and not primarily a change in larger structures. I suggest, then, that the material patterns that are involved in the second stage of learning are the result of changes of this nature, in the 10 to 40 angstrom size category. Now, how could such changes occur as a result of the operation of an electrical pattern that has been induced? Let us assume that there exist molecules, *A*, that have a definite configuration and that make up part of the nervous system, and also other molecules, *A′*, that are complementary to *A*, and that are loosely attached to the reticular framework of the brain structure near by. As Tyler has pointed out, there is evidence that mutually complementary molecules exist, perhaps in large numbers, in living organisms. Now, under the influence of an electrical pattern, there may be a change in environment in this region, a change in *p*H or in ionic concentration, which changes the equilibrium conditions for the molecules A' relative to the reticular portions of matter to which they are attached. This change in environment might permit them to dissociate away, and then to move over and to combine, because of their complementariness, with the molecules A, thus producing a material change which could itself at a later time be reflected in an electrical response.

I suppose all intramolecular forces are electrostatic in origin, inasmuch as they involve electrons and nuclei. Intramolecular forces have been classified as electronic dispersion forces, hydrogen-bond forces, interaction of ionic groups, etc.

DR. KÖHLER: If complementary configurations in your sense occur on the molecular level, and if being complementary or failing to be complementary are facts which may influence function on this level, is it not possible that analogous facts are also important on a macroscopic level?

DR. PAULING: It is very difficult to get a simple chloride ion to attach itself permanently to a structure, because thermal agitation is enough to cause it to dissociate away. You have to have larger aggregates of atoms in order to resist the effect of thermal agitation. Perhaps one of these large aggregates carries a positive charge, and its complementary structure carries a negative charge, and then when combination occurs there is a change in the electrical pattern. In a system where electrolytes are present it is not easy to consider setting up distributions of electrical charge of the order of magnitude of hundreds of angstroms, because neutralization of the charges by the electrolyte occurs, giving a double layer in which the significant dimensions are small. The idea of large-scale electrical distributions hence does not appeal to me very strongly. I prefer to think that some sort of an insulating effect is produced by these molecules, rather than that there is just an electrical pattern.

DR. KÖHLER: I asked my question because, in a discussion of memory, macroscopic field distributions in the cortex are likely to play a major role. I was wondering whether your principle can be applied to such larger configurations.

DR. PAULING: Perhaps it could, since the amount of charge separation required to produce the observed fields is very small. A pattern of loci in which charges are neutralized by complementary structures could be built up.

DR. WEISS: It may be well to point out in this connection that central neuron arrangements, even in the adult nervous system, are much more flexible and variable than we usually admit. Evidence is increasing that the nerve cell bodies react to different functional loads by changes of size. It would seem impossible that a marked increase or decrease in the size of a cell body could occur without the finer terminal connections on the cell body suffering major disruptions. There is, therefore, ample opportunity for rearrangements. We must not think of the microscopic network as something absolutely set and rigid.

DR. PAULING: There is a possibility (though it seems to me to be unlikely) that the whole phenomenon involves changes that are of a still larger dimensional order of magnitude—a thousand, five thousand, ten thousand angstroms. I would be especially interested to know what the characteristic dimensions of the structures involved are.

DR. KÖHLER: I should like to come back to the experiment which Dr. Lindsley has described. The alpha rhythm, he said, can be blocked by a visual stimulus which is too weak to be perceived. This observation seems to be at variance with Professor Adrian's interpretation of the blocking of alpha by visual stimulation. Adrian, it will be remembered, found that presentation of a homogeneous bright *field* does not disturb the rhythm very much, while even a weak *contour* may have a strong blocking effect. Since contours attract attention more than homogeneous fields do, he concluded that the rhythm is blocked by attention. But it cannot have been attention which blocked the rhythm in Dr. Lindsley's experiments. A stimulus which is not perceived will be unable to attract attention. I should therefore prefer the assumption that any inhomogeneity in the visual cortex, and in this sense a pattern, is associated with a particular process; that this process is absent, or weaker, when the visual cortex is in a homogeneous state; and that the alpha rhythm is likely to be blocked when such a pattern process is introduced.

DR. GERARD: I am afraid that it isn't that simple, Dr. Köhler. There is another bit of evidence that I am certain most men who have worked with this have experienced themselves. With a little practice, I can look directly at a 100-watt light, which is certainly not a uniform field but is patterned, and, by deliberately paying no attention to it, I can have my alpha waves remain perfectly intact; then with no change except what I can describe in no other way than as directing my attention to the light, have them immediately disappear.

DR. HALSTEAD: We became interested in the relation of vision and alpha rhythm some years ago in a search for neural concomitance of flicker fusion partly as a result of the work on photic driving reported by Adrian and Matthews in 1934. They stimulated man binocularly with intermittent light while recording the electroencephalogram from the occipital areas. The work provides samples of unilateral coupling between the frequency of the intermittent light and the alpha frequency of the EEG. A diffuse (non-patterned) light flash of relatively high intensity was employed to obtain these records—in a conscious individual. About five years ago, my associates and I were able to publish similar records of the driving of brain rhythms, with and without anesthesia, in the Macaque monkey. We obtained a good coincidence over a range from about 18 per second to about 3½ per second. This driving effect can be reproduced with reasonable predictability in several species from rabbit to man. Under the conditions of photic driving the organism becomes literally coupled to the external environment.

We also noted that if trains of flashes, especially in the neighborhood of 6 per second, were suddenly thrown onto the retinae a peculiar hump and spike pattern appeared in some animals at once and lasted from one to two seconds. This pattern is apparently indistinguishable from spontaneous brain rhythms obtained in certain forms of human epilepsy. In recent work by Walters in England, Gastaut in France, and others, petit mal seizures have been induced in man under controlled conditions in convulsive-prone individuals by this technique. The alpha rhythm is abolished for the duration of the seizure and hence may be thought of as being "blocked." However, our observations along with those of Wang, Bishop, Bartley, Toman, and others indicate clearly that the electrical responses of the normal mammalian brain from rabbit to man tend to follow or to couple with a train of light flashes through the range from about 2 flashes per second to 20 or more flashes per second. Rather than being "blocked," the most prominent brain rhythm is, under these circumstances, "paced" or "driven" by the photic stimuli. There is some evidence to be found in the literature that suggests that introduction of a contour at the periphery of a flickering field lateralizes the photic driving to the ipsilateral hemisphere. The electrical activity of the contralateral hemisphere is not "blocked," however; rather it seems to be decoupled from the flickering field.

DR. KÖHLER: Can we be quite sure that what is commonly called driving of the alpha rhythm by varying flicker frequencies actually deserves this name? We know that upon sudden illumination of the eye an on-effect can be registered from the visual cortex. Now, when the sudden illumination is repeated, on-effects may be repeated at the same rate. Have we any way of proving that in such experiments the frequency of the alpha rhythm is actually changed by rhythmic stimulation? How does one exclude the possibility that the registered waves are simply on-effects which have the frequency of the flicker?

DR. HALSTEAD: For the conditions of stimulation we employed, it is rather generally agreed that the total on-effect lasts somewhere in the neighborhood of 200 milliseconds. It is conceivable that frequencies up to about 5 per second might be reflected as trains of on-effects. As Bartley and others have shown, however, components of the on-effects are increasingly inhibited as flash frequency increases above this point.

In a favorable preparation and using a glissando technique, one may drive electrical activity continuously and without essential change in wave form from 6 or 7 per second to 20 or more per second.

Another line of evidence is afforded by the enhancement of amplitude of electrical response from the brain during photic driving. This enhancement reaches a maximum around 10 per second in monkey and man. This is generally regarded as the alpha frequency. Toman, studying these relations in man, found it possible to omit a single flash without disrupting the response. We have made similar observations. This would suggest that the centrally induced rhythm may sustain itself briefly without reinforcement.

DR. GERARD: Doesn't the rhythm continue a little while after you stop the flicker?

DR. HALSTEAD: Yes, it does; it may last from 2 to 6 seconds in some preparations.

DR. VON NEUMANN: I don't understand why sometimes even a subliminal signal of light may stop the alpha rhythm, while here it continues in spite of light signals. How are these things compatible?

DR. MC CULLOCH: Didn't you get part of an answer to your question in Dr. Gerard's statement concerning the light bulb? Whatever this mechanism is, it is important.

DR. HALSTEAD: I think that there are special circumstances under which the alpha does not block in the presence of light.

DR. KLÜVER: In this connection I should like to ask Dr. Lashley, who has described his scintillating scotomata, whether there are disturbances of the alpha rhythm during the observation of such scotomata. I am also wondering whether such disturbances, if they exist, are related to the frequency of the scintillations.

DR. MC CULLOCH: May I answer his question? I have very brilliant scintillating scotomata with hemicranial headache, or migraine. The scotomata have, inside them, an area in which I can see nothing. They begin always right next to the point of fixation, just a whisker to one side of it, and they move out until they have occupied the whole of that field. I already have several times had electrodes well placed in and about the inion and all over the back of my head and there is no change that we can detect. I don't have a good alpha, but there is no change in what alpha I have, and there are no other waves that are not normally there with my eyes open or my eyes closed.

DR. KLÜVER: What is the frequency of the scintillations per second?

DR. MC CULLOCH: The thing which I see is composed of small bright dashes and these things don't all come or go in a pulsing fashion, but they twinkle.

DR. KLÜVER: You are apparently not a good case for getting data on the frequency of oscillations. Fröhlich, by the use of a rather ingenious method, found that the frequency of oscillations in his scintillating scotomata was 20 per second.

DR. LINDSLEY: To confuse the issue further, I would like to add a comment. When I was working with infants and young children, where the alpha rhythm is of a different order of frequency, namely, around 3 or 4 per second, I found that the time for delay of the alpha rhythm in blocking is proportionately longer than it is in the adult where the frequency is around 10 per second.

DR. MC CULLOCH: There is one more piece of evidence. I don't know whether Mr. Stroud is in the audience, but if he is, I wonder if he would be willing to say something about the rate at which forms can be perceived?

MR. JOHN M. STROUD: Stanford was kind enough to let me come, as a kind of renegade physicist, into the Psychology Department and do a little work on the problem of the frequencies of scansion functions, such as the alpha rhythms (which I considered as the no-load symptom of a scanning function). The hypothesis that I wanted to test led to two experiments that are relevant here.

One was very simple. I had an ordinary cathode-ray oscilloscope and set the observer the task of finding out how fast the sweep was going, by tapping a key which put a little pip on the trace. His task was to learn to tap the key fast enough to make the pip stand still; that was his only task. It showed, incidentally, that human reactions are by no means as unprecise as one might infer from the classical reaction time experiments. Anybody could learn to tap at the rate of 10 cycles per second for one hundred consecutive taps. He could always get his reactions to fall within a band of 20 milliseconds for a prescribed frequency, with errors from one tap to the next of the order of only 2½ milliseconds.

In the second experiment the observer was set the task of matching the brightness of a flickering light by adjusting the brightness of a steady light. The field was a small circle split vertically, with the flickering light on the left and the steady, on the right. The level of illumination was low, and a very dark red light was used. The flicker was rectangular in wave form, and the proportion of the cycle during which the light was on could be controlled by the experimenter. In different runs it was varied from zero to 100 per cent. The experimenter could also vary the repetition rate from about one-half to about 20 per second.

The results varied from subject to subject, but in general showed that for frequencies above about 8 the relation between the proportion of time the flickering light was on, and brightness was linear, with a slope of 1.0. That is when the light is on 100 per cent it is matched by an equal intensity. Call this 100 per cent. Now if the flickering light is on 50 per cent of the cycle, it is matched by a steady light one-half as bright, etc.

If, however, we reduce the repetition rate below 8 per second another phenomenon appears. At 6.4 cycles for some subjects (and at lower frequencies for others) the brightness follows the previous curve for durations down to about 60 per cent. From 60 per cent to 30 per cent it remained constant, and then fell off in a linear fashion with duration.

At a still slower rate (4 per second for many subjects) the brightness dropped very slowly as the duration changed from 100 per cent to about 45 per cent. (The brightness dropped only about 10 per cent in this range.) Then it dropped rapidly as the duration was further reduced. In all cases the data seem to be best fitted by straight lines making sharp corners with one another.

The subjects often reported, where the frequencies were from 4 to 8 and the durations short, that there was a definite light and dark phase and that this was the basis for a phi movement. Most people saw the center line bulge and collapse alternately. I saw a guillotine moving up and down, chopping off the flickering half field. I found that the subject's movement hypotheses were, to a considerable extent, under their control, and that we could have all sorts of hypotheses to cover what occurred at these and at lower frequencies.

DR. MC CULLOCH: I would just like to point out that the relations here are just what one would expect from Dr. Halstead's work on the extent to which the alpha can be driven.

MR. STROUD: I know that, but I hesitated, in writing it out formally,* to call attention to its relationship to the known behavior of alpha although I was familiar with Dr. Halstead's work.

DR. BROSIN: If there are no further questions I will ask Dr. McCulloch to take care of the many that have already been posed him.

DR. MC CULLOCH: I would like, if I may, to begin at the other end, so to speak, and thank Dr. Paul Weiss for liberating me. He holds the theory, you see, that the output is in no sense to be determined by the input—it is independent. This suggestion of his makes it very easy to handle the host of questions. I cannot, for the life of me, attempt to answer them question by question. I would much rather try to give what I would call an organized statement about half a dozen major items that are under discussion.

If you will remember, I suggested that we ask the theoretical physicist to account for himself; that is, for organisms like himself, of such a nature that they could produce theoretical physics. Now, in asking that question, I did not ask that he be a psychologist, and I did not ask that he be a philosopher, and, above all, I did not ask that he be a theologian. The entire problem, I am quite sure, will never be manageable if we begin at the most difficult end. I would much rather ask, merely, that it be a theory of the physicist, and my object in doing this is to put the question concerning whether a machine of this order of complexity can state how it works. If it can (and I think it can), then we can build others that can do the same thing. My object in doing this is to keep the issues in a form in which they are manageable from the standpoint of physics and mathematics. I grew up in a medical school where my professor of neuropathology defined the subconscious as a place which is not a place where an idea is when it is not an idea, and my professor of neurology said that he knew that time was in the temporal lobe and space was in the postcentral convolution, but he was not sure where consciousness was.

Now, it is just these kinds of phrases that I would avoid. I don't believe that I brought up the question of consciousness. If I had to, in a medical sense, I would use the word only to say that this patient was or was not conscious, according to whether he could or could not bear witness to what I could also bear witness to. I would, here and now, systematically exclude any such problem. I am, however, fully aware that most of what goes on in me is something of which I am not aware; I only see the end products. I see those only rarely and in funny flashes. Very often I know that I have decided something without even knowing what I have decided. I do not believe that we have any right to ask of whatever it is that is consciousness that it do the chore of alpha waves or something else of that sort. Alpha waves as I see them (and I am coming back to them first and foremost) are a sweep of scansion. That sweep can, to some extent, be stimulus-triggered. It can be pulled down to about half; it can be pushed up to nearly double its idling rate. When we try to push it further, it snaps and gives us double frames or something else of that sort. I think that much is fairly clear. It has this peculiarity regardless of the sensory modality in which one works. One always comes to an ability to follow in the range, from, let's say, five to fifteen—the maximum number of frames that one can take in any sensory modality. The ability to pull out universals seems to be something of the order of ten per second. You can push it up a little or down a little. Therefore, whatever be the method whereby we do pull out universals, it is timed by something that runs around ten per second, and which may be pushed a little one way or the other. This means that all our ideas, insofar as they are timeless, are going to have this peculiar quality of being moments without internal temporal structure, and that our timed affairs, between our ideas, will be between them, and hence of larger temporal units. Now, that is the first point.

---

* John M. Stroud, *The Moment Function Hypothesis,* M.A. Thesis, Stanford University, 1948.

Next—as to why alpha rhythm should disappear when the instrument is in use—this has two possible answers. One is that when we go after forms—when we begin to use a device to pull out shapes—we are keying in this mechanism with incoming impulses, and in so doing we upset a sweep in that sense. But it is far more likely that what happens is that when we do use this sweep, the detail (since its voltages are in cells oriented in this same direction) will have its throw in the same direction as the apparent sweep; hence all you will get is that your sweep is passing up and down as a series of voltage fronts and your cells have their major axis in this direction, so they create voltages up and down. Therefore, you would expect that in using this structure you wouldn't see your sweep. In the present discussion I am not particularly interested in which happens.

I should add one more thing before I leave the problem of the alpha, and that is George Bishop's work on the relation of the alpha to an impulse thrown in at various times in the alpha cycle, reported in one of the Cold Spring Harbor Symposia. He showed very clearly that the response that you get in the cortex to an incoming volley is determined in large measure by the positivity or negativity of that cortex—which way up the voltage in it is at the time that the impulses arrive. There is no question but that alpha does affect the appearance of "on" and "off" effects in the superficial layers of the cortex itself, but I am not interested in any one particular mechanism at the moment. I don't care what the mechanism is. What is important to me is that we have some idea, even if it be one very hard to run down, of a process whereby we can get our "stimulus equivalence." That is the crucial item as far as I am concerned, and I am well aware that in one sense, or modality, it may go one way, and in another in another way, or, in a single modality it may pull out one thing by one method and another thing by another method. This would seem to postulate a theory of a nervous net which has delays and connections in time, and connections in space which are of such a kind that you cannot know beforehand what sort of a thing your invariant will be. It may be a figure of impulses over a single cell, or a series of cells, in time, and it may be over a flock of cells at one time. I have not tried to give a spatial picture of the nervous system or a temporal picture, but a fourfold picture—three dimensions of space and one of time in which that invariance is to be sought. That is the first thing that I want to make clear.

The second thing I want to make clear is much harder to say. I spoke of reverberating memory, and I spoke of reverberating memory to the exclusion of all other memories, not because I believe that all that we remember is so carried, or because I believe that we carry more in reverberation than in any other way, but because anything which can be done by any kind of memory can be done by a reverberating memory, except to go through periods of complete inactivity or complete explosion. As I see it, there are three problems in memory, not one, or three kinds of memory that we are going to have to distinguish always.

First, there is reverberation, and it does work. Second, there is a kind of alteration of the nervous net with use, which I believe is probably crucial in many of the experiments on conditioned behavior. It is obviously so in certain kinds of pathological situations. The third kind of memory, I don't believe could be accounted for by either of these two ways, and it is one with a bottleneck both in putting information in and in taking it out. I am utterly unhappy about where it is. I don't think that we have any better guess today than that it is somewhere in our heads. I see no particular reason for assigning it to neurons instead of to their matrix—the glia. I don't see how we can tell where we have to look as yet, because in many of the experiments in which there are lesions made in brains, we have had large amounts of territory removed. However, usually we fail to destroy most fixed memories; therefore, we cannot today locate the filing cabinets. I think that sooner or later answers to the question of those filing cabinets, or whatever it is on which is printed "photographic records" and what not, will have to be found. The theory that I set up was intended to handle only this question—it was set up to show that organisms or computing machines which digitalized, or quantized, at the level of neurons—at the level of relays—were systems capable of doing certain kinds of things, and rather a large number of things like remembering and computing any computable number.

The question as to whether I am trying to envisage the human nervous system, or that of a particular animal, or whether I'm interested only in computing machines in general is, I think, most fairly answered in this manner. Logical machines, machines for handling logical problems, can be constructed out of almost any material; however, some materials work better than other materials. I do not know out of what materials the human nervous system is constructed, but the principles of design that have to be followed, insofar as they are not matters of the material but matters of the circuit action, will be identical to the extent that the tasks they are competent to perform prescribe their structure. They need be no more, and the theory that I gave (as opposed to the particular hypotheses concerning, let's say, alpha rhythm or visual cortex) is a perfectly general theory. The hypotheses that I made relating to the nervous system are of the kind that will help me to put electrodes in one place to see what is going on in that place and to know what I am looking for, and I expect to hit a snag there very soon. I always have hit snags so far. Now, why have I chosen to quantize in nervous impulses? Well, let's say the human brain is of a general order of complexity of something like 10^{30} if we think of it in terms of its ultimate particles. One might split this at the level of the atoms, or one might split this at the level of the molecules, or one might split this at the level of the neurons, and so on. The question is: At what level can one split the behavior so as to define a set of units in terms of which to work? And, obviously, the nervous impulse at the level of the neurons is a fairly nice unit for working. It divides the complexity nearly in half. That is why I like to quantize my affairs in the behavior of the neuron and in its properties.

I look on the question of a field somewhat in this manner. In any device (whether we make it a digital device or an analogy device), the ultimate units of our universe are quantized affairs. We use it as an analogy device when we disregard the unitary composition, or definite structure of our elements, and treat them as wholes, which means that we are dealing with their parts, if you will, at best, statistically. We have, with respect to them, thrown away the information that goes into their construction, and have retained only the overall picture of their behavior. Now, I do not like to treat something like the cerebral cortex in this "field" manner. First and foremost, I will admit unquestionably that it has some statistical properties. But what I am looking for is something that will perform a logical task. I like to look for it in a thing that has a grain, and I like to take that grain as my unit, because then I can see what degrees of freedom the system has and to what extent they are bound in any given job of handling information. I believe that there are many things in the nervous system that smear the results. I worked for years on facilitation and extinction. I know that if I stimulate a small area, that there does arise between that active area and any other area, a difference in voltage which persists for a matter of minutes, and so on, with the busy area electrically negative to the depths and to remote regions. I know that if I stimulate this area and it becomes active, there is an increased blood supply; there is a shift to the acid side, and so on; and I know that these will affect, not merely the cells that are active, but (like the direct current that then or there appears) cells that lie in the region, whether or not they have themselves participated, and that this will alter the threshold of those cells.

These happen to be problems that I have worked with and I am working with now, along with the noble metal potentials or oxidation reduction potentials. I am also working on the thermal changes. All these things will smear results. They rob the system of some of its degrees of freedom without conveying information. They are the kinds of things that would arise if we had to build our electronic devices and keep them in tubs of salt water. I regard them, by and large, for my purposes, as secondary affairs to be worried about, as I worry about the isolation of the paths to ground, so that all of the grounded side of an instrument is free of troublesome voltages or currents. Now, the reason for making this distinction as sharp as possible in the face of plenty of evidence that the smearing occurs, is that we are dealing with something that has a field-like property. My reason for treating it in the manner in which I have treated it is this. It compels one so to state his theory that he can account for these changes by the digital mechanisms; that is, so that he can contrive a digital device to account for each of these. This at once increases his ability to handle his digital machine. It was clearly shown in our first paper, I believe, that these field-like processes—facilitation and extinction—could be handled by sticking in hypothetical nets, because once a system is, in this sense, quantized anywhere, it might as well be quantized everywhere. This keeps the theory clean.

The next question that I want to try to meet is one which is also very tough to state. The cortex, to my mind, is a vast computing machine. I see no reason why a deeper structure may not assign to any particular part of it a particular task today, and tomorrow assign that particular task to some other part of it—except insofar as particular portions of the cortex are stimulus-bound by a path right to them from the periphery. Those paths to it are well known now, and they include a large fraction of the total surface of the total switch board of the cortex. To the extent to which the afferent connections define a portion of the cortex as visual, it must handle something to do with vision. It must handle something to do with touch if it is the part to which touch reports. So, to the extent to which a cortex is so bound back to our sense organs, it cannot be swapped part for part. No lower mechanism can assign to the visual cortex the job of receiving impulses directly from the ears; they only go to the auditory mechanism. But, apart from that, I see no reason why the lower mechanisms may not perfectly well use one part of the cortex today and another part of the cortex tomorrow for the same function. I do doubt that it does this under ordinary circumstances, but I see no reason why, with damage to one part of the cortex, you wouldn't expect it to do just that. As a matter of fact, I think that you would expect it to do that, just as you would expect that when a man's leg is chopped off, or both legs are chopped off, the man learns to get along on his arms. It is a computing machine—it is sitting there—I see no reason why it can't use this part now and that part then.

I would like to come back now, for a moment, to the question of alpha rhythm, this time in cases of hysterical blindness. A hysterically blind man may have an alpha rhythm, and he may have the alpha rhythm with his eyes wide open, and he may have it when you present him with this, that, or the other thing to look at. He doesn't see, of course, but he reacts to it correctly in other ways; however, as far as he is concerned, he doesn't see it, and he may have a very nice alpha all of this time. Yet in the case of the next hysterically blind individual you check, it may be that the minute he opens his eyes, his alpha is gone. Now, I don't think that this hysterical blindness is something that you can pin on any one part of the system. I don't think that when you throw out the works and say, "I shall not see," that you have to do it in the visual cortex. You may, or you may do it somewhere else, so you may block out alpha or you may fail to block out alpha. And I don't think that when we deal with functions of any great order of complexity, we stand too much of a chance of finding the particular part of the cortex where that is being done, for it can be done now here, now there, except for the sensory projections upon it.

Now, let me contrast this with the answer to the second question that was asked of me, as to why was I so very much worried about the descending systems and so little concerned with the ascending systems. I am worried about the descending systems for this reason. There is no direct path from the cerebral cortex to a motor horn cell known to me. The motor horn cell whose process goes out to the muscle, if it signals to the muscle, has one and only one consequence in the muscle, but, sitting around the motor horn cells themselves, are sacs of internuncials playing around and onto the motor horn cells. To the sacs of the internuncials come stuff, information or impulses, over the dorsal roots, stuff from other parts of the spinal cord, stuff from the brain stem, and so on. The cortex, to a certain extent, tries to take over these other structures by sending signals to them along the way, but its final chore of doing anything in the periphery is achieved only by playing upon the cells which are already going according to ways of their own which are adjusted to the world over hosts of channels. Therefore, what comes out when the cortex starts doing something is by no means simply determined by what goes down from the cortex. That turns out to be actually a small contributory factor to the totality of what is playing upon the motor horn cell. Now, these lower affairs have often very persistent behavior which lasts for a matter of minutes, so that what the cortex does when it starts off something at one time, and then goes to do something else afterwards, is to have to play on systems with enormous (I want to call it inertia) tendencies to overshoot—tendencies to persist in activity. That is why I am worried about the descending system. The ascending system, for the most part, does not have this type of complexity. It has a fairly simple relay system on the way up.

Now, again, I want to make this point very clear. I'm greatly worried about the fact that at the present time we keep trying to assign things to the cerebral cortex that are being done not in it but elsewhere. The reason that we want to assign it to cortex is that if we smash cortex, we don't get it. I feel as though we tried to put the whole organism into the cortex.

The reason I want a clear theory is this. I see people who are insane or who are neurotic. I do not believe that even if you took the best psychoanalysts in the world, and they were a thousand times as numerous as they are, or are likely to become, that there would be enough of them to go around to readjust the neurotics alone, using only auditory afferents to make the adjustments. As a matter of fact, I believe that more and more, we will come to the use of interruptions of the circuit action of the nervous system by drugs, or by electrical stimulation, or by surgery; and the question is, if we want to interrupt something, where is that something happening? How can we interrupt it, and if we do interrupt it, how can we reinstate circuit action that is adaptive? That is why I want this theory, because I hope that it will guide me in placing electrodes so as to find out what is going on where.

Have I left out any crucial questions?

DR. BROSIN: I think that Dr. Gerard is in a position to see, in a judicious way, the trend of the discussion to date, and since the last three speakers have moved the locus of interest a good deal, I wonder if Dr. Gerard would give us a brief summary of what he thinks the vectors have been.

DR. GERARD: I would like to say, first, that I cannot recall attending a meeting of this sort which has gone so happily and so constructively and with such concentrated brilliance. It has been a privilege to be permitted to participate.

The main problem we are attacking is that of correlating behavior with the mechanisms for that behavior. There have been three major approaches by the speakers. There has been an examination of machines, of their basic properties and the logic concerning them, and an heroic attempt made by Dr. von Neumann to see to what extent their general properties apply to those of the nervous system, and to what extent, therefore, they might effectively guide our thinking about the mechanics of the behavior of this most complicated machine. There has been discussion of the nervous system, of the properties of neurons, of their spatial relations, their electrical attributes, and their interactions in groups. And there has been a rational approach, relating various logics to the behavior of the nervous system, which attempted, in a way, to synthesize both of the others. I couldn't help thinking, as Warren McCulloch was making his further statement just now, of how history repeats itself. Some century ago, George Boole became interested in his algebra of logic with the hope that, since the human mind made logic, a study of the laws of logic would necessarily reveal the functions of the human mind and ultimately of the brain. He was perhaps a little more naïve in his time than we are today. He was certainly not aware of, or at least did not give adequate attention to, the non-rational elements of behavior.

Dr. McCulloch just stated that he is interested in devising a theory that would account for the brain as a calculating machine. Several of the questions raised in the discussion have asked about these other aspects. What about the non-rational side? What about the urge for the machine to calculate? What about the drive, so striking in some of the men in this symposium, which, perhaps even more than the elegance of the calculating machine, determines whether or not something worth while comes out of the system in the course of its lifetime? If one wishes to allocate those other aspects to a different box than the cortex, that is perhaps permissible, but it does not resolve the problems that seem to remain.

With calculating, there come inevitably the problems of learning and memory. There has been much discussion of them, and I hesitate to introduce, at this time, any new factual material; but it might be interesting again to make a glissando over the various types of memory that have been suggested. I am always suspicious when I find myself putting things into sharp compartments and saying, "This is this, and that is that." I don't think nature loves a wall. Some experiments have been reported, by Reynolds particularly, on a type of memory in the nervous system which seems intermediate between the reverberating type and the permanent type. At least, if it be reverberating, it is certainly over far longer times that we ordinarily associate with reverberation. He came to it from problems of pain and relates it to causalgia.

Symmetrical teeth with symmetrical defects in a person's jaw were given dental treatment. On one side the filling was done with great care against trauma and pain, on the other side, with a minimum of this desirable care. In nearly all those patients, severe referred pain developed on the traumatized side. This could be elicited routinely by pricking the opening to the antrum of the maxilla. The sensitivity and referred pain lasted for months, but was immediately and irreversibly abolished by a brief period of conduction block of the nerve from that tooth. Some kind of central overactivity, of inflammation if you will, was able to persist for long periods of time apparently under minimal peripheral reinforcement.

As to the mechanisms for establishing these important behavior attributes, it seems to me that we have wandered in our discussion over a number of hierarchies—the molecular, the neuronal, the cerebral—and the question has arisen whether we need something beyond—the man or the observer—to know what is happening in all of those. This last question can safely be left to the remaining periods of the symposium.

We all agree, I believe, that molecular events are critical in determining neuronal behavior. We all agree that neuronal events are critical in determining the total behavior of the nervous system itself. The discussion has focused mainly on this step. Dr. McCulloch has explored it from the particular viewpoint of the nervous system as a digital machine, functioning exclusively in terms of quanta, of the nerve impulses which are or are not discharged by particular neurons under particular conditions. Others have emphasized other kinds of relationships, of the analogical type. The real question being asked, I believe, is whether, in addition to a rigid hierarchy of interaction from molecular to neuronal to total performance, there can also be a direct influence from the molecular level to the molar level.

$$\begin{tikzcd}
\text{Molecular} \arrow[r] \arrow[rr, bend left, "?"] & \text{Neuronal} \arrow[r] & \text{Cortical (C.N.S.)} \arrow[r] & \text{Man}
\end{tikzcd}$$

This question is also involved in the one of whether neurons can interact with each other to form some sort of total functioning unit by mechanisms in addition to the simple quantal discharge of elements.

At the one extreme, the position could be taken that all that happens in the nervous system depends on digital machines and unitary discharges. At the other extreme, the position could be taken that these are rather trivial in the functioning of the nervous system, having to do with the more routine activities controlling movement and the like; but that all the more complicated functions depend on analogical behavior, involving chemicals, electrical fields, spontaneous changes, which might act in many other ways than as a scansion mechanism. Some might say that these are the power determinants of the more complicated types of behavior. I sincerely doubt, however, that anyone in the room would take either of those extreme positions. Rather, we are all prepared to recognize that both elements enter into the picture. The point of disagreement would certainly be about the relative importance of the various mechanisms and their relative significance in particular aspects of total behavior. That is as far as I should go at this time.

I was a bit shocked by Dr. McCulloch's comment, a moment ago, that all the other phenomena are merely degradations in the functioning of a beautiful digital mechanism. I really don't think that he believes that, and perhaps I misunderstood; for the very alpha rhythm that he uses as his scanning device is non-digital. In any event, the speakers who are to follow will surely emphasize these other aspects, the mass action effects and the potential fields, and we have already had a considerable attention to the chemical aspects, which likewise transcend the simple matter of discharge of impulses along neurons. We can confidently look forward to equally stimulating and provocative periods in the remainder of the symposium.

DR. VON NEUMANN: I would like to bring up something that I consider remarkable and something which I think did not come out very explicitly except in one remark of Dr. McCulloch's, and I would like to emphasize it. I think that in regard to the great flexibility, there are few symptoms—if one thinks of the functioning of the nervous system and compares it with the functioning of very complicated automata—which are very conspicuous in the case of the nervous system and very conspicuously absent in any automaton that we know of, which may be easily related with each other and related with the circumstances which one ought to expect to emerge at this level of complication. It is very obvious that the brain differs from all artificial automata that we know; for instance, in the ability to reconstruct itself (as in the case of mechanical damage). It is always characterized by a very great flexibility in the sense that animals which look reasonably alike and do the same thing, may do it by rather different cerebral mechanisms. Furthermore, though all humans belong to the same category and do the same things, outwardly, in some cases they are using different cerebral mechanisms for the same things, so there seems to be a flexibility of pathways.

There are some indications (I do not wish to go into them in detail now) that some parts of the organism can act antagonistically to each other, and in evolution it sometimes has more the character of a hostile invasion of one region by another than of evolution proper. I believe that these things have something to do with each other and also with the following. The largest automaton which we know how to plan could consist of something of the order of 10^4 units, and this is already very complicated. By this, I mean the fact that they are still nominally made from blueprints, and, in all cases where an automaton is built, there exists a blueprint for it, and there is somebody around to guarantee that it was built from the blueprint. These things are, however, very marginal. It is probably not true for most automata of this degree of complexity that they are really identical copies of what is on the blueprints. And it is probably no longer even true that there is anybody around who had anything except a somewhat intuitive relation to it. This is increasingly true for complicated machines. No amount of time and skill and reliability in components will change this in an automaton more complicated than this, and they will probably become more and more complicated. There are a hundred thousand to a million parts involved in some of them, and this is still low, compared to the brain. Building will probably be a completely nominal operation, not completely controlled by what one finds in blueprints. It will have a different relation to the object, and by that relation, I mean this. It has already happened (and it is, of course, just by the introduction of automata into mathematics that it begins to happen) that you are no longer thinking about the subject, but thinking about an automaton which would handle the subject. It has already happened in the introduction of mass production into industry that you are no longer producing the product, but you are producing something which will produce the product. The cut is, at present, never quite sharp, and we still maintain some kind of relation with the ultimate thing that we want. Probably the relationship is getting looser. It is not unlikely that if you had to build an automaton now you would plan the automaton, not directly, but on some general principles which concern it, plus a machine which can put these into effect, and will construct the ultimate automaton and do it in such a way that you yourself don't know any more what the automaton will be. You will do it in such a way that all you will know is what you want to do by it, and by what qualitative methods it can be done, and how to make an automaton which can put this into effect. But as to how the primary thing does it, you need not know. You don't need to know what the result of every particular multiplication was which occurred in the process of solving the equation. If you come to such a principle of construction, all that you need plan and understand in detail is the primary automaton, and what you must furnish to it is a rather vaguely defined matrix of units; for instance, 10^{10} neurons which swim around in the cortex. But the thing becomes functioning only by being organized by the primary thing, and it is only the primary thing that you need plan. Then you begin to have this trait; you do not simply build a primary machine to build a secondary one and then separate them. If you do not separate them but they stay in contact with each other, and if the functioning of the secondary machine presupposes that its contact is a primary one, then, I think that it is achievable that the thing can be watched by the primary automaton and be continuously reorganized when the need arises. I think that if the primary automaton functions in parallel, if it has various parts which may have to act simultaneously and independently on separate features, you may even get symptoms of conflict. Also, through that, since the secondary matrix is introducing things which are probably not rigorously defined in all details, the same primary automaton in two cases may not produce exactly the same structure. And, if the planning is well done, there will probably be a very high probability that the secondary thing will function the same way in all cases; but there will not be certainty, and, if you concentrate on marginal effects, you may observe the ensuing ambiguities. I just wanted you to realize that though these notions are vague, they indicate phenomena of this character. Especially when you go to much higher levels of complexity, it is not unreasonable to expect symptoms of this kind.

The Problem of Serial Order in Behavior

K. S. LASHLEY
Harvard University and the Yerkes Laboratories
of Primate Biology

The previous speakers have approached our common problem by considering the properties of the elementary units of which we believe the cerebral structure to be built up. They have considered the kinds of neural integration or behavior which can be anticipated from those properties. The remaining members of the symposium have in their research been concerned chiefly with the analysis of complex behavior, seeking to derive general principles of neural integration from the infinitely complex products of that integration. Our common meeting ground is the faith to which we all subscribe, I believe, that the phenomena of behavior and of mind are ultimately describable in the concepts of the mathematical and physical sciences. In my discussion here, I have deliberately turned to the opposite extreme from the neuron and have chosen as a topic, one aspect of the most complex type of behavior that I know; the logical and orderly arrangement of thought and action. Our discussion so far has dealt chiefly with the conditions of input and of immediate switching in the nervous mechanism, without explicit consideration of what is already going on within the system.

My principal thesis today will be that the input is never into a quiescent or static system, but always into a system which is already actively excited and organized. In the intact organism, behavior is the result of interaction of this background of excitation with input from any designated stimulus. Only when we can state the general characteristics of this background of excitation, can we understand the effects of a given input.

The unpronounceable Cree Indian word "kekawewechetushekamikowanowow" is analyzed by Chamberlain (7) into the verbal root, *tusheka,* "to remain," and the various particles which modify it as follows: *ke(la)wow,* the first and last syllables, indicating second person plural; *ka,* a prefix of the future tense; *we,* a sort of imperative mode expressing a wish; *weche,* indicating conjunction of subject and object; *mik,* a suffix bringing the verb into agreement with a third person subject and second person object; and *owan,* a suffix indicating that the subject is inanimate and the object animate. A literal translation: "You will I wish together remain he-you it-man you" or, freely, "may it remain with you." This difference in structure between Cree and English illustrates an outstanding characteristic of verbal behavior; the occurrence of predetermined, orderly sequences of action which are unique for each language. In English the adjective precedes, in French it follows the noun which it modifies. In English the movement or action of the subject is expressed as early as possible after the subject; in German the expression of action may be postponed until all qualifying thoughts have been expressed. In a sentence discussing this subject, Pick (20) introduces fifty-five words between the subject and the principal verb. Each Chinese word, and to a lesser extent, each English word, stands as an unchanging unit. In the highly inflective languages, such as Sioux, the form of almost every word in the sentence may be altered, according to some attribute of the subject, as when two objects rather than one or several are discussed.

The study of comparative grammar is not the most direct approach to the physiology of the cerebral cortex, yet Fournié (10) has written, "Speech is the only window through which the physiologist can view the cerebral life." Certainly language presents in a most striking form the integrative functions that are characteristic of the cerebral cortex and that reach their highest development in human thought processes. Temporal integration is not found exclusively in language; the coordination of leg movements in insects, the song of birds, the control of trotting and pacing in a gaited horse, the rat running the maze, the architect designing a house, and the carpenter sawing a board present a problem of sequences of action which cannot be explained in terms of successions of external stimuli.

ASSOCIATIVE CHAIN THEORIES

In spite of the ubiquity of the problem, there have been almost no attempts to develop physiological theories to meet it. In fact, except among a relatively small group of students of aphasia, who have had to face questions of agrammatism, the problem has been largely ignored. It is not even mentioned in recent textbooks on neurophysiology or physiological psychology, nor is there any significant body of experimental studies bearing upon the problem. The spinal animal scarcely exhibits serial activity, so the physiologist may be excused for overlooking the phenomenon. On the other hand, psychologists have been concerned chiefly with the question of whether or not the organizing processes displayed in serial action are conscious, and very little with the organization itself. I have chosen to discuss the problem of temporal integration here, not with the expectation of offering a satisfactory physiological theory to account for it, but because it seems to me to be both the most important and also the most neglected problem of cerebral physiology. Temporally integrated actions do occur even among insects, but they do not reach any degree of complexity until the appearance of the cerebral cortex. They are especially characteristic of human behavior and contribute as much as does any single factor to the superiority of man's intelligence. A clearer formulation of the physiological problems which they raise should be of value, even though a solution of the problems is not yet in sight.

I shall consider first some of the questions raised by the structure of language, then turn to other forms of serial action for indications of the nature of the nervous mechanisms involved.

To the best of my knowledge, the only strictly physiological theory that has been explicitly formulated to account for temporal integration is that which postulates chains of reflexes, in which the performance of each element of the series provides excitation of the next. This conception underlay the "motor theories" of thinking which were advocated by several psychologists early in this century. Watson (26) sought to identify thought with inaudible movements of the vocal organs, linked together in associative chains. The peripheral chain theory of language was developed in greatest detail by Washburn (25). She distinguished what she called "successive movement systems" and, although she drew her examples from memorized series of nonsense syllables, her implication was that such series are typical of all language behavior. She defined a movement system as "a combination of movements so linked together that the stimulus furnished by the actual performance of certain movements is required to bring about other movements." She described speech as a succession of vocal acts in which the kinesthetic impulses from each movement serve as a unique stimulus for the next in the series (25, pages 11 ff.). Attempts to confirm these peripheral theories by mechanical (Thorsen, 23) or electrical (Max, 19) recording of muscular tensions have given no valid evidence in support of them. It should be noted that, at the time when the theories were proposed, it was generally believed that conduction in the nervous system is always downstream from sense organ to muscle, and that muscular contraction must always follow promptly on stimulation. The existence of reverberatory circuits which could maintain central activity was scarcely suspected.

The introspective psychology which objected to such peripheral theories did not explicitly formulate an alternative neurological theory, but there is implicit in it a view that verbal thought is a simple chain of central processes in which each element serves to arouse the next by direct association. Titchener, for example, maintained that the meaning of a word (or of an auditory image in his system) consists of the chain of associations which it arouses; that it has no meaning until such a sequence has occurred. From this it must be inferred that he was thinking in terms of a simple associative chain, since no other relating process is suggested.

OBJECTIONS TO THE ASSOCIATIVE CHAIN THEORY

A consideration of the structure of the sentence and of other motor sequences will show, I believe, that such interpretations of temporal organization are untenable and that there are, behind the overtly expressed sequences, a multiplicity of integrative processes which can only be inferred from the final results of their activity. There is an extensive controversial literature dealing with this inferred integrative activity. Pick (20) devotes almost his entire book, *Die agrammatischen Sprachstörungen*, to reviewing discussions of the subject. Most of this literature deals with the question of whether or not the integrative processes are conscious. Much of this is irrelevant to the present topic, but the advocates of so-called imageless thought did present a great deal of material indicative of the complexity of the problem of thought structure. From this, and other evidence which I shall present, I believe that the production of speech involves the interaction of at least three, possibly four, major neurological systems which are interrelated but somewhat independently variable.

Let us start the analysis of the process with the enunciation of the word. Pronunciation of the word "right" consists first of retraction and elevation of the tongue, expiration of air and activation of the vocal cords; second, depression of the tongue and jaw; third, elevation of the tongue to touch the dental ridge, stopping of vocalization, and forceful expiration of air with depression of the tongue and jaw. These movements have no intrinsic order of association. Pronunciation of the word "tire" involves the same motor elements in reverse order. Such movements occur in all permutations. The order must therefore be imposed upon the motor elements by some organization other than direct associative connections between them. So, for the individual movements in writing or typing the word, finger strokes occur in all sorts of combinations. No single letter invariably follows *g*, and whether *gh*, *ga*, or *gu* is written depends upon a set for a larger unit of action, the word.

Words stand in relation to the sentence as letters do to the word; the words themselves have no intrinsic temporal "valence." The word "right," for example, is noun, adjective, adverb, and verb, and has four spellings and at least ten meanings. In such a sentence as "The mill-wright on my right thinks it right that some conventional rite should symbolize the right of every man to write as he pleases," word arrangement is obviously not due to any direct associations of the word "right" itself with other words, but to meanings which are determined by some broader relations.

It has been found in studies of memorization of nonsense syllables that each syllable in the series has associations, not only with adjacent words in the series, but also with more remote words. The words in the sentence have, of course, associations with more remote words as well as with adjacent ones. However, the combination of such direct associations will not account for grammatical structure. The different positions of the word "right" in the illustrative sentence are determined by the meanings which the positions in relation to other words denote, but those meanings are given by other associations than those with the words in the spoken sentence. The word can take its position only when the particular one of its ten meanings becomes dominant. This dominance is not inherent in the words themselves.

From such considerations, it is certain that any theory of grammatical form which ascribes it to direct associative linkage of the words of the sentence overlooks the essential structure of speech. The individual items of the temporal series do not in themselves have a temporal "valence" in their associative connections with other elements. The order is imposed by some other agent.

This is true not only of language, but of all skilled movements or successions of movement. In the gaits of a horse, trotting, pacing, and single footing involve essentially the same pattern of muscular contraction in the individual legs. The gait is imposed by some mechanism in addition to the direct relations of reciprocal innervation among the sensory-motor centers of the legs. The order in which the fingers of the musician fall on the keys or fingerboard is determined by the signature of the composition; this gives a *set* which is not inherent in the association of the individual movements.

THE DETERMINING TENDENCY

What then determines the order? The answer which seems most in accord with common sense is that the intention to act or the idea to be expressed determines the sequence. There are, however, serious difficulties for this solution. There is not much agreement among psychologists concerning the nature of the idea. The structuralist school, under the leadership of Titchener, held that the idea consists of mental images, often the auditory images of words, and the meanings are nothing but sequences of such images. Describing the role of images in his lecturing, Titchener wrote (24), "When there is any difficulty in exposition, a point to be argued *pro* and *con* or a conclusion to be brought out from the convergence of several lines of proof, I hear my own voice speaking just ahead of me." What solution of the lecture problem for the lazy man! He need not think but only listen to his own inner voice; to the chain of associated auditory images. A behaviorist colleague once remarked to me that he had reached a stage where he could arise before an audience, turn his mouth loose, and go to sleep. He believed in the peripheral chain theory of language. (This clearly demonstrates the superiority of behavioristic over introspective psychology. The behaviorist does not even have to listen to his own inner voice.)

Seriously, such positions offer no solution for the problem of temporal integration. Titchener finds his grammar ready made and does not even raise the question of the origin of the succession of images. The chain-reflex theory, while definite, is untenable.

The third view of the nature of the idea was developed by a group known as the "Würzburg School" (see Boring, 4); exponents of imageless thought. It held that some organization precedes any expression that can be discovered by introspective or objective means. Thought is neither muscular contraction nor image, but can only be inferred as a "determining tendency." At most, it is discovered as a vague feeling of pregnancy, of being about to have an idea, a Bewustseinslage. It is not identical with the words which are spoken, for quite frequently no word can be recalled which satisfactorily expresses the thought, and we search a dictionary of synonyms until a word or phrase is found which does seem appropriate.

In his discussion of the relation of thought to speech, Pick (20) accepts this point of view, but he asserts further that the set or the idea does not have a temporal order; that all of its elements are cotemporal. Evidence in support of this conclusion comes, for example, from translation of one language into another which has a different sentence structure. I read a German sentence, pronouncing the German words with no thought of their English equivalents. I then give a free translation in English, without remembering a single word of the German text. Somewhere between the reading and free translation, the German sentence is condensed, the word order reversed, and expanded again into the different temporal order of English. According to Epstein (9), the polyglot shifts readily from one language to another, expressing the same thought in either, without literal translation. The readiness with which the form of expression of an idea can be changed, the facility with which different word orders may be utilized to express the same thought, thus is further evidence that the temporal integration is not inherent in the preliminary organization of the idea.

THE SCHEMA OF ORDER

The remaining alternative is that the mechanism which determines the serial activation of the motor units is relatively independent, both of the motor units and of the thought structure. Supporting evidence for this may be found in the mistakes of order, the slips and interferences which occur in writing and speaking. For some time I have kept records of errors in typing. A frequent error is the misplacing or the doubling of a letter. *These* is typed t-h-s-e-s, *look* as l-o-k-k, *ill* as i-i-l. Sometimes the set to repeat may be displaced by several words. The order is dissociated from the idea. Earlier, in preparing this paper, I wrote the phrase, "maintain central activities." I typed *min*, omitting the *a*, canceled this out and started again; *ama*. The impulse to insert the *a* now dominated the order. I struck out the *a* and completed the phrase, only to find that I had now also dropped the *a* from *activities*. This example suggests something of the complexity of the forces which are at play in the determination of serial order and the way in which conflicting impulses may distort the order, although the primary determining tendency, the idea, remains the same.

The polyglot, who has become proficient in a secondary language, who thinks in it and even dreams in it, may still tend to use the grammatical structure of his native tongue. If, as in French, that tongue applies gender to inanimate things, the English pronouns referring to them may take the gender of the French equivalents, though the French nouns are not thought. The German postponement of the verb or the Magyar use of the past infinitive may be incorporated in the new language. In such cases, the structuring seems to be dissociated both from the content and from the simple associative connections of the words themselves.

The ease with which a new structure may be imposed on words is illustrated by the quickness with which children learn hog Latin. The form which I learned involved transposing the initial sound of each word to the end of the word and adding a long *a*. Thus—at-thay an-may oes-gay own-day e-thay eet-stray. Some children become very facile at such inversions of words, and re-structure new words without hesitation. From such considerations it seems to follow that syntax is not inherent in the words employed or in the idea to be expressed. It is a generalized pattern imposed upon the specific acts as they occur.

"PRIMING" OF EXPRESSIVE UNITS

There are indications that, prior to the internal or overt enunciation of the sentence, an aggregate of word units is partially activated or readied. Evidence for this comes also from "contaminations" of speech and writing. The most frequent typing errors are those of anticipation; the inclusion in the word being typed of some part of a word or word structure which should properly occur later in the sentence. It may be only a letter. Thus I wrote, *wrapid* writing, carrying the *w* from the second word to the first. Not infrequently words are introduced which should occur much later in the sentence, often five or six words in advance.

In oral speech, Spoonerisms illustrate the same kind of contamination. The Spoonerism is most frequently an inversion of subject and object: "Let us always remember that waste makes haste." But it may be only a transposition of parts of the words: "Our queer old dean" for "our dear old queen." The frequency with which such contaminations occur is increased by haste, by distraction, by emotional tension, or by uncertainty and conflict as to the best form of expression. In some types of aphasia the tendency to disordered arrangement of words is greatly increased, and, in extreme cases, the attempt to speak results in a word hash with complete loss of grammatical organization. Professor Spooner, after whom such slips are named, was probably suffering from a mild form of aphasia. In these contaminations, it is as if the aggregate of words were in a state of partial excitation, held in check by the requirements of grammatical structure, but ready to activate the final common path, if the effectiveness of this check is in any way interfered with.

In his *Psychopathology of Everyday Life*, Freud has given numerous examples of similar contaminations of action outside the sphere of language. We do not need to accept his theories of censorship and suppression to account for such slips. They are of the same order as misplacements in typing and represent contaminations of co-existing, determining tendencies to action.

Such contaminations might be ascribed to differences in the relative strength of associative bonds between the elements of the act, and thus not evidence for pre-excitation of the elements or for simultaneous pre-excitation. However, the understanding of speech involves essentially the same problems as the production of speech and definitely demands the postulation of an after-effect or after-discharge of the sensory components for a significant time following stimulation. Thus, in the spoken sentence, "Rapid righting with his uninjured hand saved from loss the contents of the capsized canoe," the associations which give meaning to righting are not activated for at least 3 to 5 seconds after hearing the word.* I shall refer later to other evidence for such long after-discharge of sensory excitations. The fact of continued activation or after-discharge of receptive elements and their integration during this activation justifies the assumption of a similar process during motor organization. The processes of comprehension and production of speech have too much in common to depend on wholly different mechanisms.

INTERNAL AND OVERT SPEECH

One other point with respect to the organization of speech: The earlier literature on aphasia emphasized the distinction of internal and overt speech. The aphemia of Broca and the pure motor aphasia of Wernicke and later writers were held to be a loss of the ability to enunciate without loss of ability to think in words and without paralysis of the organs of speech. The brain insult was assumed to affect only the transition from the thought to the enunciation of the word. We may doubt the existence of instances of such "pure" defects and question the reliability of the early clinical examinations in view of the more careful analyses that have been made since 1917, but the distinction of internal and overt speech is still valid and the transition still unexplained. Watson interpreted internal speech as inaudible movements of the vocal organs, and Jacobsen (15) and Max (19) have given evidence of changes in muscular tonus during verbal thinking or thought of movement. This is far from proving that the motor discharge is essential for the internal formation of words, however.

I once devised an instrument to record small movements of the tongue. Within the limits of its sensitivity, it showed that in silent thinking the tongue usually drops to the back of the mouth and shows no detectable movement. Verbal problems, such as the correct squaring of three-place numbers, could be carried out with no trace of overt movement. If, however, I urged the subject to hurry or if I slapped his face, his tongue came forward and showed movements corresponding to the syllabification of internal speech or of the computation he was performing. This I interpret as indicating that internal speech may be carried out wholly by processes within the nervous system, with some unessential discharge upon the final common path for vocal movements. Facilitation of the motor path, either by increased emotional tension or by "voluntary" reinforcement, increases its excitability until the same central circuits whose activity constitutes internal speech are able to excite the overt movements. This aspect of the language function is irrelevant to the problem of syntax or serial order, but is important as illustrating a further point in the dynamics of the cerebrum. Many activities seem to require for their performance both a specific patterning and also a general facilitation, a rise in dynamic level. There are, I think, indications that hemiplegia and motor aphasia are primarily expressions of a low level of facilitation rather than a loss of specific integrative connections which are involved in the use of language or in the patterning of our movements. A monkey, for example, after ablation of the precentral gyrus may seem unable to use the arm at all, but if emotional excitement is raised above a certain level, the arm is freely used. As soon as the excitement dies down, the arm is again hemiplegic. I have seen something of the same sort in a human hemiplegic. The problem of the availability of memories, which was raised earlier in the discussion here, may find a partial solution in such fluctuations in dynamic level. In many of the organic amnesias the pattern of integration seems to be retained but can be reactivated only by an abnormally intense sensory or central reinforcement.

---

* Dr. Lashley ingeniously laid the groundwork for this three paragraphs earlier, when he mentions "wrapid writing." The audience all heard, "Rapid writing with his uninjured hand," etc. "Capsized canoe" required a complete and amusing about-face. EDITOR.

GENERALITY OF THE PROBLEM OF SYNTAX

I have devoted so much time to discussion of the problem of syntax, not only because language is one of the most important products of human cerebral action, but also because the problems raised by the organization of language seem to me to be characteristic of almost all other cerebral activity. There is a series of hierarchies of organization; the order of vocal movements in pronouncing the word, the order of words in the sentence, the order of sentences in the paragraph, the rational order of paragraphs in a discourse. Not only speech, but all skilled acts seem to involve the same problems of serial ordering, even down to the temporal coordination of muscular contractions in such a movement as reaching and grasping. Analysis of the nervous mechanisms underlying order in the more primitive acts may contribute ultimately to the solution even of the physiology of logic.

It is possible to designate, that is, to point to specific examples of, the phenomena of the syntax of movement that require explanation, although those phenomena cannot be clearly defined. A real definition would be a long step toward solution of the problem. There are at least three sets of events to be accounted for. First, the activation of the expressive elements (the individual words or adaptive acts) which do not contain the temporal relations. Second, the determining tendency, the set, or idea. This masquerades under many names in contemporary psychology, but is, in every case, an inference from the restriction of behavior within definite limits. Third, the syntax of the act, which can be described as an habitual order or mode of relating the expressive elements; a generalized pattern or schema of integration which may be imposed upon a wide range and a wide variety of specific acts. This is the essential problem of serial order; the existence of generalized schemata of action which determine the sequence of specific acts, acts which in themselves or in their associations seem to have no temporal valence.

I shall turn now to other phenomena of movement which may be more readily phrased in physiological terms and which may suggest some of the mechanisms underlying serial order.

DURATION AND INTENSITY OF NERVOUS DISCHARGE

A consideration of the control of extent and rate of movement supports the view that sensory factors play a minor part in regulating the intensity and duration of nervous discharge; that a series of movements is not a chain of sensory-motor reactions. The theory of control of movement which was dominant at the turn of the century assumed that, after a movement is initiated, it is continued until stopped by sensations of movement and position, which indicate that the limb has reached the desired position. This theory was opposed by a good bit of indirect evidence, such as that accuracy of movement is increased rather than diminished with speed. I had opportunity to study a patient who had a complete anesthesia for movements of the knee joint, as a result of a gunshot wound of the cord (16). In spite of the anesthesia, he was able to control the extent and speed of movements of flexion and extension of the knee quite as accurately as can a normal person.

The performance of very quick movements also indicates their independence of current control. "Whip-snapping" movements of the hand can be regulated accurately in extent, yet the entire movement, from initiation to completion, requires less than the reaction time for tactile or kinesthetic stimulation of the arm, which is about one-eighth of a second, even when no discrimination is involved. Such facts force the conclusion that an effector mechanism can be pre-set or primed to discharge at a given intensity or for a given duration, in independence of any sensory controls.

CENTRAL CONTROL OF MOTOR PATTERNS

This independence of sensory controls is true not only of intensity and duration of contraction of a synergic muscle group but is true also of the initiation and timing of contraction of the different muscles in a complex movement. The hand may describe a circular movement involving coordinated contractions of the muscles of the shoulder, elbow, and wrist in about $\frac{1}{10}$ second, and the stopping of movement at a given position, of course, is only a small fraction of that time. The finger strokes of a musician may reach sixteen per second in passages which call for a definite and changing order of successive finger movements. The succession of movements is too quick even for visual reaction time. In rapid sight reading it is impossible to read the individual notes of an arpeggio. The notes must be seen in groups, and it is actually easier to read chords seen simultaneously and to translate them into temporal sequence than to read successive notes in the arpeggio as usually written.

Sensory control of movement seems to be ruled out in such acts. They require the postulation of some central nervous mechanism which fires with predetermined intensity and duration or activates different muscles in predetermined order. This mechanism might be represented by a chain of effector neurons, linked together by internuncials to produce successive delays in firing. In some systems the order of action may be determined by such a leader or pace-setter. Buddenbrock (6) has shown for the stick insect, and Bethe (3) for a number of animals from the centipede to the dog, that removal of one or more legs results in a spontaneous change in the order of stepping. Thus, for the insects, the normal order is alternate stepping of the first pair of legs with right first, left second, right third leg advancing together. With removal of the left first leg, the right first and left second alternate and the order becomes right first, left third, right third stepping together, with left second and right second advancing together, instead of alternately. These investigators were interested in spontaneity of reorganization, rather than in the mechanism of coordination, and did not propose any theory for the latter. They did show, however, that it is necessary to remove the leg completely to get the change in pattern of movement; sensory impulses from a limb stump would prevent it. Such coordination might be explained, perhaps, by a combination of loss of excitability in the centers of the absent limb, by the excitation of the remaining anterior center as a leader or pace-setter, and the spread of alternate waves of inhibition and excitation from the more anterior to the more posterior limb centers. The spontaneous change in coordination shows, however, that the coordination is not due to the action of predetermined anatomic paths but is the result of the current physiological state of the various limb centers.

Such an hypothesis implies also the assumption of a polarization of conduction along the neuraxis, with the order of excitation determined by the spatial arrangement of the centers of the legs. I see no other possibility of accounting for the facts. The examples of circular movement and of finger coordination, involving temporal integration of movements, seem to call for a similar hypothesis. They might be ascribed to an habitual linkage of the movements through a simple chain of internuncials but for two facts. First, such series are usually reversible at any point or can be started from any point. This would require the assumption of a second set of internuncials habituated to conduct in the opposite direction, and this in turn leads to the further assumption of a polarization of conduction. Second, such patterns of coordinated movement may often be transferred directly to other motor systems than the ones practiced. In such transfer, as to the left hand for writing, an analysis of the movements shows that there is not a reduplication of the muscular patterns on the two sides, but a reproduction of movements in relation to the space coordinates of the body. Try upside-down mirror writing with the left hand and with eyes closed for evidence of this. The associative linkage is not of specific movements but of directions of movement. An analysis of systems of space coordinates suggests mechanisms which may contribute to production of such series of movements in a spatial pattern.

SPACE COORDINATE SYSTEMS

The work of Sherrington, Magnus, and others on postural tonus and reflexes has defined one level of spatial integration rather fully, yet it is doubtful if these studies have revealed the effective neural mechanism. The work has shown that the tonic discharge to every muscle in the postural system is influenced by afferent impulses from every other muscle, toward increased or decreased activity, according to its synergic or antergic action. To these influences are added vestibular and cerebellar effects. Diagrammatically these mutual influences of the muscular system may be represented by separate reflex circuits from each receptor to every muscle, as Sherrington (21, p. 148) has done. But no neuro-anatomist would, I am sure, maintain that such separate circuits or paths exist. What the experiments on posture actually show is a correlation of sensory stimulation and of tonic changes in a network of neurons whose interconnections are still undefined. The reactions isolated experimentally have the characteristics of simple directly conducted reflexes, but their combination results in patterns of movement and posture which have definite relations to the axes of the body and to gravity.

This postural system is based on excitations from proprioceptors. The distance receptors impose an additional set of space coordinates upon the postural system, which in turn continually modifies the coordinates of the distance receptors. The dropped cat rights itself, if either the eyes or the vestibular senses are intact, but not in the absence of both. The direction of movement on the retina imposes a directional orientation on the postural system. Conversely, the gravitational system imposes an orientation on the visual field. Upright objects such as trees or the corners of a room appear upright, at no matter what angle the head is inclined. Derangement of the vestibular system can disturb the distance orientation or the orientation of the receptors, as in the apparent swaying of the vertical as a result of the after-images of motion following hours of rocking in a small boat.

There are other, still more generalized systems of space coordinates. We usually keep track of the compass points or of some more definite index of direction by a temporal summation of the turns made in walking, though not always with success. Finally, there is a still more plastic system in which the concepts of spatial relations can be voluntarily reversed, as when one plays blindfold chess alternately from either side of the board.

Explanation of these activities, these complex interactions, in terms of simple isolated interconnections of all of the sensory and motor elements involved seems quite improbable on anatomic grounds and is ruled out by results of our experiments on sectioning of the spinal cord. Ingebritzen (14) studied rats with double hemisection of the cord; one-half of the cord cut at the second, the other at the fifth cervical segment. In the best case only a small strand of the spinocerebellar tract of one side remained intact. These rats were able to balance in walking, oriented to visual stimuli, scratched with the right or left hind foot according to the side of the face stimulated, were able to run mazes correctly, and even learned to rise on the hind feet and push down a lever with the forepaws in opening a box.

The alternative to the isolated-path theory of the space coordinates is that the various impulses which modify postural tonus are poured into a continuous network of neurons, where their summated action results in a sort of polarization of the entire system. I shall consider later the integrative properties of such a net. For the moment I wish to emphasize only the existence of these systems of space coordinates. Their influences pervade the motor system so that every gross movement of limbs or body is made with reference to the space system. The perceptions from the distance receptors, vision, hearing, and touch are also constantly modified and referred to the same space coordinates. The stimulus is *there*, in a definite place; it has definite relation to the position of the body, and it shifts with respect to the sense organ but not with respect to the general orientation, with changes in body posture.

Memories of objects usually give them position in the space system, and even more abstract concepts may have definite spatial reference. Thus, for many people, the cardinal numbers have definite positions on a spiral or other complicated figure. What, if anything, such space characters can contribute to temporal integration is an open question. They provide a possible basis for some serial actions through interaction of postural and timing mechanisms.

RHYTHMIC ACTION

The simplest of the timing mechanisms are those controlling rhythmic activity. T. Graham Brown (5) first showed by his studies of deafferented preparations that the rhythmic movements of respiration and progression are independent of peripheral stimulation and are maintained by a central nervous mechanism of reciprocal innervation. He suggested that this mechanism of reciprocal innervation, rather than the simple reflex, is the unit of organization of the whole nervous system. He thus foreshadowed, in a way, the conception of reverberatory circuits which is coming to play so large a part in neurological theory today. Holst (13) has recently shown that the rhythmic movement of the dorsal fin of fishes is a compound of two superimposed rhythms, that of its own innervation and that of the pectoral fins. These two rhythms are centrally maintained.

Musical rhythms seem to be an elaboration of the same sort of thing. The time or beat is started and maintained at some definite rate, say 160 per minute. This rate is then imposed upon various activities. The fingers of the musician fall in multiples of the basic rate. If the leader of a quartet speeds up the time or retards, all the movements of the players change in rate accordingly. Not only the time of initiation but also the rate of movement is affected. The violinist, in a passage requiring the whole bow, will draw the bow from frog to tip at a uniform rate for the required number of beats, whether the tempo is fast or slow. With practiced violinists, the rate of movement is extremely accurate and comes out on the beat at the exact tip of the bow.

Superimposed on this primary rhythm is a secondary one of emphasis, giving the character of 3/4, 4/4, 6/4, or other time. The mechanism of these rhythms can be simply conceived as the spread of excitation from some centers organized for reciprocal innervation; as a combination of the principles of Brown and of Holst. There are, however, still more complicated rhythms in all music. That of the melodic line is most uniform. In much music, the melodic progression changes in 2, 4, or some multiple of 4 measures. In improvisation, the performer keeps no count of measures, yet comes out almost invariably in a resolution to the tonic of the key after some multiple of eight measures. Here a generalized pattern is impressed on the sequence, but it is a simpler pattern than that of grammatical structure. It only requires the recurrence of a pattern at certain rhythmic intervals; a pick-up of a specific pattern after so many timed intervals.

There are, in addition, still less regular rhythms of phrasing and emphasis. Parallels to these can be found in speech. The skilled extemporaneous speaker rounds his phrases and speaks with a definite though not regular rhythm.

The rhythms tend to spread to almost every other concurrent activity. One falls into step with a band, tends to breathe, and even to speak in time with the rhythm. The all pervasiveness of the rhythmic discharge is shown by the great difficulty of learning to maintain two rhythms at once, as in three against four with the two hands. The points to be emphasized here are the widespread effects of a rhythmic discharge indicating the involvement of almost the entire effector system, the concurrent action of different rhythmic systems, and the imposition of the rate upon both the initiation and speed of movement. Consideration of rhythmic activity and of spatial orientation forces the conclusion, I believe, that there exist in the nervous organization, elaborate systems of interrelated neurons capable of imposing certain types of integration upon a large number of widely spaced effector elements; in the one case transmitting temporally spaced waves of facilitative excitation to all effector elements; in the other imparting a directional polarization to both receptor and effector elements. These systems are in constant action. They form a sort of substratum upon which other activity is built. They contribute to every perception and to every integrated movement.

INTERACTION OF TEMPORAL AND SPATIAL SYSTEMS

Integration ascribed to the spatial distribution of excitations in the nervous system has been much more intensively studied than the temporal aspects of nervous activity. Theories of integration are based almost exclusively upon space properties, time entering only in theories of facilitation, inhibition, and after-discharge. In cerebral functions, however, it is difficult to distinguish between spatial and temporal functions. The eye is the only organ that gives simultaneous information concerning space in any detail. The shape of an object impressed on the skin can scarcely be detected from simultaneous pressure, but the same shape can readily be distinguished by touch when traced on the skin with a moving point or when explored by tactile scanning. The temporal sequence is readily translated into a spatial concept. Even for vision it might be questioned whether simultaneous stimulation gives rise directly to space concepts. The visual object is generally surveyed by eye movements, and its form is a reconstruction from such a series of excitations. Even with tachistoscopic exposures, the after-discharge permits a temporal survey, and, with visual fixation, shifts of attention provide an effective scanning.

Since memory traces are, we believe, in large part static and persist simultaneously, it must be assumed that they are spatially differentiated. Nevertheless, reproductive memory appears almost invariably as a temporal sequence, either as a succession of words or of acts. Even descriptions of visual imagery (the supposed simultaneous reproductive memory in sensory terms) are generally descriptions of sequences, of temporal reconstructions from very fragmentary and questionable visual elements. Spatial and temporal order thus appear to be almost completely interchangeable in cerebral action. The translation from the spatial distribution of memory traces to temporal sequence seems to be a fundamental aspect of the problem of serial order.

I spoke earlier of the probability of a partial activation or priming of aggregates of words before the sentence is actually formulated from them. There is a great deal of evidence for such preliminary facilitation of patterns of action in studies of reaction time and of word association. Reaction time, in general, is reduced by preliminary warning or by instructions which allow the subject to prepare for the specific act required. In controlled association experiments, the subject is instructed to respond to the stimulus word by a word having a certain type of relation to it, such as the opposite or a part of which the stimulus is the whole; black-white, apple-seed. The result is an attitude or set which causes that particular category to dominate the associative reaction. Whether such preliminary reinforcement is to be ascribed to accumulation of excitatory state, as defined by Sherrington (21), or to some other physiological process, the facts of behavior assure that it is a genuine phenomenon and plays a decisive role in determining the character of the response.

Once the existence of such states of partial activation is recognized, their possible role in temporal integration must be considered. There are indications that one neural system may be held in this state of partial excitation while it is scanned by another. Here is an example. A series of four to six numbers is heard: 3–7–2–9–4. This is within the attention or memory span and is almost certainly not remembered in the sense in which one's telephone number is remembered, for memory of it is immediately wiped out by a succeeding series of numbers. While it is retained in this unstable way, subject to retroactive inhibition, the order of the numbers can be reassorted: 3–7–2–9–4, 3–2–7–9–4, 4–9–2–7–3, and the like. It is as if, in this case, a rhythmic alternation can suppress alternate items, or a direction of arousal can be applied to the partially excited system. Another example which illustrates even more clearly the spatial characteristics of many memory traces is the method of comultiplication, used in rapid mental calculation. In attempts to play a melody backward, we have a further illustration. I find that I can do it only by visualizing the music spatially and then reading it backward. I cannot auditorily transform even "Yankee Doodle" into its inverse without some such process, but it is possible to get a spatial representation of the melody and then to scan the spatial representation. The scanning of a spatial arrangement seems definitely to determine, in such cases, the order of procedure. Two assumptions are implied by this. First, the assumption is that the memory traces are associated, not only with other memory traces, but also with the system of space coordinates. By this I do not mean that the engram has a definite location in the brain; our experiments show conclusively that such is not the case. Rather, when the memory trace is formed it is integrated with directional characters of the space system, which give it position in reference to other associated traces. Second, the assumption is that these space characters of the memory trace can be scanned by some other level of the coordinating system and so transformed into succession.

This is as far as I have been able to go toward a theory of serial order in action. Obviously, it is inadequate. The assumptions concerning spatial representation and temporal representation may even beg the question, since no one can say whether spatial or temporal order is primary. Furthermore, such determining tendencies as the relation of attribute to object, which gives the order of adjective and noun, do not seem to be analyzable into any sort of spatial structure or for that matter, into any consistent relationship. I have tried a number of assumptions concerning the selective mechanism of grammatical form (spatial relations, the relative intensity or prominence of different words in the idea, and so on) but I have never been able to make an hypothesis which was consistent with any large number of sentence structures. Nevertheless, the indications which I have cited, that elements of the sentence are readied or partially activated before the order is imposed upon them in expression, suggest that some scanning mechanism must be at play in regulating their temporal sequence. The real problem, however, is the nature of the selective mechanism by which the particular acts are picked out in this scanning process, and to this problem I have no answer.

Such speculations concerning temporal and spatial systems do little more than illustrate a point of view concerning nervous organization which is, I believe, more consistent both with what is known of the histology and elementary physiology of the brain and also with behavior phenomena than are the more widely current theories of simple associative chains of reactions.

Nearly forty years ago Becher (2, page 243) wrote: "There is no physiological hypothesis which can explain the origin and relations of temporal forms in mental life; indeed, there is no hypothesis which even foreshadows the possibility of such an explanation." The situation is little better today, but I do feel that changing conceptions of the fundamental organization of the nervous system offer more hope for a solution of such problems than did the physiological knowledge available when Becher wrote. However, we are still very far from being able to form an explicit explanation of temporal structure.

THE FUNDAMENTAL MECHANISM OF INTEGRATION

Neurological theory has been dominated by the belief that the neurons of the central nervous system are in an inactive or resting state for the greater part of the time; that they are linked in relatively isolated conditioned reflex arcs and that they are activated only when the particular reactions for which they are specifically associated are called out. Such a view is incompatible both with the widespread effects of stimulation which can be demonstrated by changes in tonus and also with recent evidence from electrical recording of nervous activity. It is now practically certain that all the cells of the cerebrospinal axis are being continually bombarded by nerve impulses from various sources and are firing regularly, probably even during sleep. The nervous activity which they in turn elicit depends upon the current physiological state of the neurons with which they are connected. It is probably not far from the truth to say that every nerve cell of the cerebral cortex is involved in thousands of different reactions. The cortex must be regarded as a great network of reverberatory circuits, constantly active. A new stimulus, reaching such a system, does not excite an isolated reflex path but must produce widespread changes in the pattern of excitation throughout a whole system of already interacting neurons.

The facts of cerebral structure support such a view. The cortex is composed chiefly of neurons with short axons. LeGros Clark (8) has found for the striate area of the monkey that Marchi degeneration extends for only a short distance from a point of injury. In the striate area of the rat, I have never been able to trace degeneration beyond three or four cell diameters from the margin of a lesion, and I believe that this lack of long transcortical fibers is true of other areas as well as of the visual cortex. Visual perception reveals close integration of different parts of the striate areas in spite of the absence of long association fibers. In the visual cortex of the rat there are only 19 neurons for each afferent fiber. To produce the animal's visual acuity, all of the afferent fibers must be firing continually. There are approximately 34,000 cell bodies in the lateral geniculate nucleus of the rat, and the minimum number of visual units necessary to produce the visual acuity of the rat is actually above this figure. (The acuity is determined by direct experimental tests.) These figures should be of interest in relation to the numerical values cited by Dr. von Neumann. The number of cells in the visual cortex of the rat is only about 10^6, and in some of my experiments where I have removed the greater part of the visual cortex the capacity for discrimination of visual forms has been retained when no more than 20,000 cells of the visual cortex remain. There is also evidence that no part of the cerebral cortex except the visual areas is essential for visual perception and memory.

DR. LORENTE DE NÓ: What is the number of afferents in the optic nerve?

DR. LASHLEY: There are 290,000 afferents in the optic nerve of the rat, and the figure is reduced to 34,000 in the lateral geniculate. The actual numbers are 9,000,000 myoids, 290,000 ganglion cells, and 34,000 cells in the lateral geniculate. That may include cells with short axons also. There are about 125,000 cells in each of the five layers of the cortex. These figures are for one eye and hemisphere.

DR. VON NEUMANN: In the human being the corresponding number is about 125,000,000 for the first, isn't it?

DR. LASHLEY: I know of no figure for that level.

DR. LORENTE DE NÓ: It hasn't been analyzed in any way.

DR. VON NEUMANN: The optic nerve corresponds to the second one?

DR. LASHLEY: Yes. The axons of the ganglion cells pass through the optic nerves. There is an average of 300 visual cells firing into each central pathway. There are fewer than 5 cells in the receptive layer of the visual cortex of the rat for each afferent fiber of the optic radiation and only 19 cells per afferent fiber in the entire visual cortex. Since the visual acuity of the rat requires that all of the 34,000 cells of the radiation be firing constantly, it seems certain that all of the neurons within the striate areas, the visual cortex, must be firing constantly. There is a good bit of evidence that all of the integrative functions of vision are carried out within the striate areas. In the rat, I have removed, from one or another animal, practically every other part of the isocortex without disturbing visual perception or memory. With monkeys I have removed the supposed visual associative areas without producing any significant loss of visual functions.

These facts lead to the conclusion that the same cells in the visual cortex participate in a great variety of activities. Practically all of the cells of the area must be fired by every visual stimulation, and these same cells must be the ones which retain the visual memories. The conclusion follows that differential responses depend upon the pattern of cells which are excited in combination. The visual cortex is a network of cells of short axon without long interconnections between its parts or with other cortical areas. Its integrative functions are an expression of the properties of such a network.

The same conception must be applied to other cortical areas. There are, of course, long association tracts in the cortex, such as the corpus callosum, the superior longitudinal fasciculus, and the temporo-frontal tracts. Once, 26 years ago, I suggested facetiously that these might be only skeletal structures, since I could find no function for them. No important functions of these tracts have yet been demonstrated. Section of the corpus callosum produces only a slight slowing of reaction time, ipsilateral as well as contralateral (Akelaitis, 1); section of occipito-frontal fibers produces, perhaps, a temporary disturbance of visual attention but no other symptoms. The integrative functions seem to be carried out as well without as with the main associative tracts. The major integrative functions must, therefore, be carried out by the network of cells of short axon. The properties of such networks of cells must be analyzed before the mechanisms of the cerebral cortex can be understood. Something can be inferred from the characteristics of excitability of cells and their arrangement in recurrent loops. If, as seems a necessary conclusion from the histology of the striate area, all of the cells of the network are subject to constant excitation and are firing whenever they recover from the refractory state, then mutual interference of circuits will produce complicated patterns throughout the area, patterns which will stabilize in the absence of differential stimulation, as is perhaps indicated by the regularity of the alpha rhythm. Any new afferent impulses reaching the area can only produce a reorganization of the existing pattern. What happens at any particular point in the system, as at an efferent neuron, is the statistical outcome of the interaction of myriads of neurons, not of the transmission of impulses over a restricted path, of which that efferent cell forms a link. It is possible to isolate parts of the system by operative means or by anesthetics and so to get a one-to-one relation of stimulus locus and responding muscles, from which the reflex mechanism has been inferred. As Goldstein (12) has pointed out, however, the parts isolated in the reflex are influenced by a multiplicity of effects in the intact organism of which there is little or no trace in the isolated preparation.

I can best illustrate this conception of nervous action by picturing the brain as the surface of a lake. The prevailing breeze carries small ripples in its direction, the basic polarity of the system. Varying gusts set up crossing systems of waves, which do not destroy the first ripples, but modify their form, a second level in the system of space coordinates. A tossing log with its own period of submersion sends out periodic bursts of ripples, a temporal rhythm. The bow wave of a speeding boat momentarily sweeps over the surface, seems to obliterate the smaller waves yet leaves them unchanged by its passing, the transient effect of a strong stimulus. Wave motion is not an adequate analogy because the medium which conveys the waves is uniform, whereas the nerve cells have their individual characteristics of transmission which at every point may alter the character of the transmitted pattern.

The great number of axon terminations on every nerve cell has not been considered in theories of integration. It implies, of course, that the cell can be fired by impulses from a variety of sources. But it also suggests another possibility, more fruitful for understanding of integrative processes. A nerve impulse arriving over a single axon terminal may not fire the cell but may modify its excitability to impulses from other sources. In an elaborate system of neurons such subthreshold effects might establish a pattern of facilitation which would determine the combination of cells fired by subsequent excitations. The space coordinate system and various types of *set* or priming may be pictured as patterns of subthreshold facilitation pervading the network of neurons which is activated by the more specific external stimulus.

Such a view of the mechanism of nervous action certainly does not simplify the problems nor does it as yet provide any clue to the structuring that constitutes the set or determining tendency, or to the nature of such relations as are implied in the attribute-object, opposites, or other abstract concepts. A few relations seem reducible to spatial terms, part-whole, for example, but even for these there is no clear conception of the neural basis of their space properties. These considerations do not, I believe, contradict fundamentally the basic conceptions that have been formulated by Dr. McCulloch. They do, however, indicate a direction of necessary elaboration. The nets active in rhythmic and spatial organization are apparently almost coextensive with the nervous system. The analysis must be extended to the properties of such nets; the way in which they are broken up into reactive patterns in the spread of excitation, to give, for example, directional propagation or its equivalent. I strongly suspect that many phenomena of generalization, both sensory and conceptual, are products, not of simple switching, but of interaction of complex patterns of organization within such systems.

SUMMARY

The problems of the syntax of action are far removed from anything which we can study by direct physiological methods today, yet in attempting to formulate a physiology of the cerebral cortex we cannot ignore them. Serial order is typical of the problems raised by cerebral activity; few, if any, of the problems are simpler or promise easier solution. We can, perhaps, postpone the fatal day when we must face them, by saying that they are too complex for present analysis, but there is danger here of constructing a false picture of those processes that we believe to be simpler. I am coming more and more to the conviction that the rudiments of every human behavioral mechanism will be found far down in the evolutionary scale and also represented even in primitive activities of the nervous system. If there exist, in human cerebral action, processes which seem fundamentally different or inexplicable in terms of our present construct of the elementary physiology of integration, then it is probable that that construct is incomplete or mistaken, even for the levels of behavior to which it is applied.

In spite of its present inadequacy, I feel that the point of view which I have sketched here holds some promise of a better understanding of cerebral integration. Attempts to express cerebral function in terms of the concepts of the reflex arc, or of associated chains of neurons, seem to me doomed to failure because they start with the assumption of a static nervous system. Every bit of evidence available indicates a dynamic, constantly active system, or, rather, a composite of many interacting systems, which I have tried to illustrate at a primitive level by rhythm and the space coordinates. Only when methods of analysis of such systems have been devised will there be progress toward understanding of the physiology of the cerebral cortex.

REFERENCES

1. Akelaitis, A. J. Studies on the corpus callosum. ii. The higher visual functions in each hononymous field following complete section of the corpus callosum. *Arch. Neurol. Psychiat.*, 1941, 45, 788–796.
2. Becher, E. *Gehirn und Seele.* Heidelberg, 1911.
3. Bethe, A. Plastizität und Zentrenlehre. *Handb. d. norm. u. path. Physiol.*, 1931, 15 (zweite H.), 1175–1220.
4. Boring, E. G. *A history of experimental psychology.* New York: Century Co., 1929.
5. Brown, T. G. On the nature of the fundamental activity of the nervous centers. *J. Physiol.*, 1914, 48, 18–46.
6. Buddenbrock, W. v. Die Rhythmus der Schreitbewegungen der Stabheuschrecke Dyxippus. *Biol. Centralb.*, 1921, 41, 41–48.
7. Chamberlain, A. F. Indians, North American. *Enc. Brit.*, 1911, 14, 452–482.
8. Clark, W. E. LeGros. Observations on the associative fiber system of the visual cortex and the central representation of the retina. *J. Anat. London*, 1941, 75, 225–236.
9. Epstein, I. *La pensée et la polyglossie.* Paris: Payot et Cie (no date).
10. Fournié. *Essai de psychologie.* Paris, 1887.
11. Fritsch, G., and Hitzig, E. Ueber die elektrische Erregbarkeit des Grosshirns. *Arc. f. Anat. u. Physiol.*, 1870, pp. 300–332.
12. Goldstein, K. *The organism.* Boston: Ginn & Co., 1939.
13. Holst, N. v. Vom Wesen der Ordnung im Zentralnervensystem. *Die Naturwissenschaften,* 1937, 25, 625–631; 641–647.
14. Ingebritzen, O. C. Coordinating mechanisms of the spinal cord. *Genet. Psychol. Monogr.*, 1933, 13, 483–555.
15. Jacobsen, E. Electrophysiology of mental activities. *Amer. J. Psychol.*, 1932, 44, 677–694.
16. Lashley, K. S. The accuracy of movement in the absence of excitation from the moving organ. *Amer. J. Physiol.*, 1917, 43, 169–194.

17. Lashley, K. S. The mechanism of vision. xvii. Autonomy of the visual cortex. *J. Genet. Psychol.*, 1942, **60**, 197–221.
18. Lashley, K. S. The mechanism of vision. xviii. Effects of destroying the visual "associative areas" in the monkey. *Genet. Psychol. Monogr.*, 1948, 37, 107–166.
19. Max, L. W. Experimental study of the motor theory of consciousness. IV. *J. Comp. Psychol.*, 1937, **24**, 301–344.
20. Pick, A. *Die agrammatischen Sprachstörungen*. Berlin, 1913.
21. Sherrington, C. S. *The integrative action of the nervous system*. London: Constable, 1906.
22. Sherrington, C. S. Some functional problems attaching to convergence. *Proc. Roy. Soc.*, B, 1929, **105**, 332–362.
23. Thorsen, A. M. The relation of tongue movements to internal speech. *J. Exp. Psychol.*, 1925, 8, 1–32.
24. Titchener, E. B. *Lectures on the experimental psychology of the thought processes*. New York: The Macmillan Co., 1909.
25. Washburn, M. F. *Movement and mental imagery*. Boston: Houghton Mifflin, 1916.
26. Watson, J. B. Is thinking merely the action of the language mechanisms? *Brit. J. Psychol.*, 1920, **11**, 86–104.

DISCUSSION

DR. KLÜVER: In 1914, there appeared a book which, in the opinion of the author, marked an epoch—since during the remaining years of his life he celebrated the day the manuscript was completed instead of his birthday. The author was von Monakow, and the title of the book was *Die Lokalisation im Grosshirn*. There is a remarkable connection between von Monakow's monumental contribution to neurology in 1914 and Dr. Lashley's presentation today. In my opinion, this is the first time since 1914 that a neurological thinker has presented such a trenchant analysis of the role of the time factor in behavior. If you study von Monakow's book you find that the central concept of his work is the concept of a "chronogenic localization," a concept which has been almost completely ignored during the intervening decades.

It is not possible today to enter into a discussion of the numerous problems raised by Dr. Lashley's presentation. As regards the relation of thinking to temporal organization, we are, it seems to me, confronted with a certain dilemma. Ideas, concepts, and meanings themselves have no reference to time and space, and, yet, the expression, formulation, and identification of ideas are processes proceeding in time and occurring in space. It is the relation of ideas to temporal and spatial factors which is of interest to the physiological psychologist. Even the fact that the verbal expression of an idea is linked up with a temporal sequence of acoustic events raises intricate questions. I got an inkling of some of the difficulties on a visit to Vogt's Institute for Brain Research, where an investigator utilized the methods of experimental phonetics in studying the speech utterances of neurological and psychiatric patients. He found, on the one hand, that the curves he had obtained could not be interpreted without considering the acoustic events "carriers of meanings" and, on the other hand, that certain forms of expression survived speeding up and slowing down and even the complete reversal of the temporal sequence. I do not know whether any further attempts have been made to utilize the techniques of experimental phonetics.

I should like to comment on only one other point. It is true that the same idea may be expressed in various languages by utilizing a different temporal order of words, just as the same perceptual relation may be recognized on the basis of different relata; but it is also true that the structure of language may prevent us from expressing ideas and thinking along certain lines. The fact that the language I am using now does not possess a word for a rough, wet object undergoing a color change may have the consequence that I overlook such an object or that I do not identify, remember, and think about it. Consequently, such an item may not or cannot enter a temporal sequence utilized in expressing an idea. In this connection Allers' experiments on "word nearness" and "word remoteness" are of particular interest. According to Allers, every attempt to express an idea in words induces a tendency towards selecting items in terms of word nearness. Word nearness and word remoteness may become decisive in determining the temporal sequence and in expressing or not expressing an idea. The line of demarcation between word-near and word-remote elements is subject to marked variations and even varies from individual to individual. No doubt, the sequence of phenomena calling for an expression in words, the sequence of words, and the sequence of ideas are sequences of a different order. The great value of Dr. Lashley's analysis lies in the fact that it exhibits the significant factors involved in the expression of ideas as well as in other instances of serial ordering, and that it utilizes such factors effectively in formulating mechanisms of cerebral activity.

DR. KÖHLER: Dr. Lashley rightly said that human orientation in space is mostly achieved in a sequence of events. We move our eyes, or our attention wanders, and so forth, as we inform ourselves about our environment. Curiously enough, however, while the process of becoming so informed occurs in time, and consists of successive acts, the result appears almost independent of this temporally extended origin. Apart from objective changes, which we may ignore in this connection, the space in which we live, and the objects around us, appear for the most part perfectly stationary. Dr. Lashley agrees, of course, with this observation. I wonder whether he can tell us how in this case the history of becoming informed disappears so completely from the final information.

DR. GERARD: I tried yesterday, in a brief summary, to remain strictly non-partisan while indicating the various viewpoints that had been presented and which were still to come. Actually, I find it impossible to think through or even towards the complexities of behavior if restricted to atomic units traveling along atomic fibers. Even the work we did on the frog brain, which showed a potential field present in which moving waves of electrical activity could travel and cross complete anatomical cuts, seems inexplicable at that level. My own inclination is to think of the discrete elements and the patterns of their activity as involved in the more particular performances of the nervous system, in the immediate sequences of action, as emphasized, but also as active in the more integrated behavior of the whole of the nervous system. This doesn't mean that, in any way, I am disagreeing with Dr. Lashley's final comment. One can hope, with some confidence, that all the properties of the most complex nervous system will prove to be adumbrated in the properties of the simplest element and connection when we know those properties sufficiently well.

I would like to raise one particular point. Some years ago, Culler and his colleagues reported an observation which may have been elaborated or may have been refuted; I haven't been following that literature. If it is correct, it seems very relevant to this whole problem, and I'd like Dr. Lashley's reaction to it. In dogs, conditioned in some particular way, a circumscribed region of the cortex, not in the area either of the receptor or of the effector, became electrically excitable and produced the conditioned response. When the conditioned reflex was extinguished, that area became inexcitable. There was, thus, a sharp localization of conditioning in that particular case, one of the few striking exceptions to the more general pattern of mass activity. What is the present status of such work?

DR. HALSTEAD: I couldn't help thinking, as I listened to this excellent example of what I would call the higher mental processes, that I could have prevented all this by putting a lesion in a particular part of one nervous system—the anterior portion of Dr. Lashley's prefrontal areas. If instead I had put a lesion in area 19 of Dr. Lashley, certain important elements in his presentation would undoubtedly have been lacking. Yet we could have removed probably each lobe of the brain individually without disturbing the basic form or organization.

I have been greatly impressed with the case that Dr. Lashley has made for non-specific, non-mosaic representation. I would like to emphasize, and if I am incorrect I hope that he will point it out, that his view is not incompatible with progressive specialization of function. The language functions that he talked about are not disturbed, except when particular areas in the brain are involved. The visual functions that he described are not disturbed, except when particular regions of the brain are involved. The important thing is that, within the region, there seems to be the possibility for equivalence to arise.

DR. LORENTE DE NÓ: It is typical of Dr. Lashley that he places on the board a series of figures that no anatomist had ever determined. Dr. Lashley had to determine them himself in order to go on with his work.

I don't think that I have yet fully appreciated the value of Dr. Lashley's talk. I will have to read it and study it thoroughly, but, while I was listening, there was going through my head a mental picture of the future development of a number of experiments that I intend to perform—suggested to me by Dr. Lashley's speech. One of the problems that Dr. Lashley has suggested to me is very accessible of experimentation in the study and treatment of things of purely central origin. There is one very good situation in which we meet discharges of very long duration, which can be created in the absence of every peripheral influence. You know that stimulation of the labyrinth produces a rhythmical movement of the eye and establishes the fact that this rhythm is not dependent upon the feedback from the eye muscles for its production. Impulses can be recorded directly from the nerves to the eye muscles, and therefore any sensory feedback is eliminated. What the labyrinth sends to the nervous system is a continuous stream of impulses, not interrupted rhythmically. The interruptions are produced in the nervous system, and, furthermore, can be produced when the labyrinths are extirpated. Thus in the usual vector nystagmus, if one labyrinth is extirpated, the spontaneous nystagmus will last some 10 or 12 or 15 days after that, and then the spontaneous nystagmus ceases. If the second labyrinth is then extirpated, the nystagmus reappears and lasts for 7 or 8 days. This is a purely central phenomenon —there are no impulses of any kind coming from the periphery into the centers, and the nystagmus can be recorded from the motor nerves after the extirpation of the eye. Consequently, it is describable as rhythmical activity, and is the type of rhythm that I'm going to investigate as soon as I get to it.

I would also like to make a remark in reference to Dr. Gerard's statement about neural elements in cerebral physiology. We have been, for many years, considering only the transmission of impulses as the natural mode. Now, this potential field of yours is another mechanism by which the nerve cells act upon others. Sometimes neurons act upon others by the transmission of impulses, and sometimes by the potential field of the currents that they establish.

DR. WEISS: The great value of Dr. Lashley's presentation lies in the fact that it places rigorous limitations upon the free flight of our fancy in designing models of the nervous system, for no model of the nervous system can be true unless it incorporates the properties here described for the real nervous system. You will recognize that our current models are far short of satisfying this condition.

Dr. Lashley's theses receive crucial support from the student of development. The embryologist has long been up against a tremendous task. While the physiologist and psychologist deal with the ready-made machine of the nervous system and can add to it as many properties as he thinks necessary, the embryologist must explain just how such an immensely intricate, yet orderly, thing can develop. These studies are still in their infancy, but a few things have already come out which gibe completely with the conclusions that Dr. Lashley has presented here; for instance, the relative autonomy of structured patterns of activity, and the hierarchical principle of their organization. As I said previously, the nervous system is not one big monotonic pool whose elements can be freely recombined in any number of groupings, thereby giving an infinite variety of nervous responses. This used to be the old idea of the associationists, and it is utterly incompatible with what we have learned about the development of the nervous system and its functions in animals. I wouldn't have spoken here if Dr. Lashley hadn't said that he is convinced that whatever happens in the brain of man is foreshadowed, and, in principle, is the same as what happens in the lower organisms. Therefore, assuming that premise, I would now like to add a few comments.

First, we know from the lower organisms that the working of the central nervous system is a hierarchic affair in which functions at the higher levels do not deal directly with the ultimate structural units, such as neurons or motor units, but operate by activating lower patterns that have their own relatively autonomous structural unity. The same is then true for the sensory input, which does not project itself down to the last final path of motor neurons, but operates by affecting, distorting, and somehow modifying the pre-existing, preformed patterns of central coordination, which, in turn, then confer their distortions upon the lower patterns of effection and so on. The final output is then the outcome of this hierarchical passing down of distortions and modifications of intrinsically preformed patterns of excitation, which are in no way replicas of the input. The structure of the input does not produce the structure of the output, but merely modifies intrinsic nervous activities that have a structural organization of their own. This has been proved by observation and experiment. Coghill has shown that the motor patterns of the animal develop prior to the development of sensory innervation. I have shown, as others have, that the removal of the sensory innervation does not abolish the coordination of motor activities. Moreover, coordinated motor functions of limbs and other parts develop even if these parts have been experimentally prevented from ever becoming innervated by sensory fibers. Therefore, the sensory pathway can have nothing to do with the structure of the motor response. There are still some authors who try to save the old associationist idea that actually the input shapes the structure of the output. I think that they are fighting a losing fight, and I think that today's discussion ought to have given them the coup de grâce. The essential independence of the structure of motor activity is dramatically demonstrated when one exchanges and reverses the limbs of animals and then finds them crawling backwards whenever they aim to crawl forwards, and vice versa. Many of you have seen my film where this had been done in the developed animal, but the same operations have been done in embryos and these animals have then functioned in reverse from the very beginning. What more spectacular expression can there be of the intrinsic primacy of the motor patterns of behavior for which the external input acts only as a selective trigger?

The autonomous development of central functions raises a further question: Are the response patterns preformed as merely static conditions, or are they of dynamic nature, that is, properties of automatic central activities? Lashley has favored the latter view, and, again, experiments in lower animals furnish corroborative evidence. Intrinsic automatic rhythms have been shown, for instance, by Adrian in the brain stem of the goldfish and in insect ganglia, by Prosser in other arthropods, by Bremer and by von Holst in the spinal cord, and by Bethe in jellyfish. I have shown experimentally that any group of bulbar or spinal nerve cells taken from vertebrates, if deprived of their structural bonds of restraining influences and allowed to undergo a certain degree of degradation, will display permanent automatic, rhythmic, synchronized activity of remarkable regularity. Rhythmic activity, therefore, seems a basic property of pools of nervous elements. Whether this automatism is generated by neuron switch works or rather by the rhythmic waxing and waning of metabolic activity expressing itself in the electric field, as suggested by Dr. Gerard, is a secondary question. The principal point is that the rhythm is not something generated through an input rhythm, but is itself a primary rhythm which may be released and even speeded up or retarded by the input, but is not derived from the input. So we have experimental evidence that autonomy of pattern, rhythmic automatism, and hierarchical organization are primary attributes of even the simplest nervous systems, and I think that this unifies our view of the nervous system. I conclude with expressing my hope that today's discussion will really mark a turning point in the building of neurological theories so that more regard will be given to all the actual facts that we do know about nervous systems.

DR. MC CULLOCH: There is a paper by Wiener, written when he was working with Bertrand Russell, in which he described the space of implications. It is a peculiarly degenerate sort of space. Now I, like many other men, find it difficult to think in motion. When I think about motion, I freeze it in a four-space. If you take Wiener's degenerate space of implication and make yourself a four-space image of it, you can visualize these rhythms as fixed forms. You can practically superimpose large numbers of these forms easily in the mind instead of battling with two or three. That is the first point, and it is merely a technical device.

Second, you remember that I said that on the way down from the cerebral cortex I was utterly unhappy about our knowledge of how the cortex did this or that on account of our ignorance of the lower mechanisms. I want to say a word about two rhythmical affairs that we are now working on. If, in the intact animal, you stimulate the nucleus caudatus, the animal just slumps. If, however, you cut bilaterally the cerebral peduncles, through which all impulses coming from the cerebrum have to go to reach the hindbrain and lower structures, and if you then stimulate the nucleus caudatus, you get beautiful automatically associated rhythmical movements. One, for example, resembles a cat fishing in the gold-fish bowl. Another resembles feline boxing. There is always some part of the body, arm, leg, or face, leading in each of these movements. The sequence of each movement belonging to this group starts in the axial system, then goes to the axio-appendicular, and thence to the appendicular, moving ever outward. I have never seen any evidence of somatotopic localization in the input to the nucleus caudatus. Yet here, in its output, on coming down any one puncture, stimulation at superior positions gives responses beginning in the tail and hindleg, deeper, in the forelegs, and near the very bottom, in the face. From the bottom you get complicated lapping and chewing movements just as you do from the amygdala. You get similar movements from the putamen under these circumstances and always this representational reversal, the lowest part being represented highest. Now, if you go down their descending systems, whether you go down the ansa lenticularis or go into the bunch of fibers that pass into the subthalamus and turns down, you can follow right down, stimulating them with similar results. Let me remind you that your stimulus is just a series of impulses, say 40 or 30 per second. The frequency of the movements increases with the voltage of stimulation, to a maximum less than 10 per second. You continue to get these rhythmical movements as you proceed downstream until you reach the pneumotaxic center, when, instead of other movements, panting supervenes. Downstream of the pneumotaxic center, just as you get only torsion to the right or else to the left, so you get only inhalation or else exhalation, or only facilitation or else relaxation. Now the interesting thing is that these rhythmical movements increase in frequency as you increase the voltage of stimulation up to a limiting value of nearly 10 per second. Above that, you never get them, for hypertonus begins to come into the background and stops the movement.

This system, whatever else it does, takes a stream of impulses from the basal ganglia, which is merely a continuous stream, over more or fewer parallel paths and produces rhythmical performances. Thus, whatever makes the rhythm is downstairs; it need not be in the cerebral cortex or in the basal ganglia. These rhythmical movements originate downstream. By destruction upstream, you may release the same thing.

DR. LINDSLEY: Dr. Lashley's discussion of the temporal schema imposed upon the motor system, was of considerable interest to me. I don't know whether the spontaneous rhythms of the cortex have any relationship to this temporal schema, but there are a good many suggestive things that we can pick up in a rather superficial look at cortical activity. We can get, through the electroencephalogram, things which have been rather impressive to me. No one has been able to demonstrate conclusively that any of the cortical rhythms of spontaneous character has a direct relationship to the resting tremors of the normal, or, for that matter, the neurologically abnormal person, despite the fact that their frequencies may be similar in range. However, in the brain waves of new-born infants and even of fetal infants, it is possible to demonstrate rhythms, particularly in the motor area of the cortex, and they seem to be at least grossly correlated with some of the serial and sequential orders of motor activity. The correlation must remain a very rough one at the present state, because we have rather inadequate techniques for investigation.

The frequency that one finds in the fetus or in the new-born infant over the motor areas of the brain is much slower than that in the adult stage. Whether this has any relation to the similar lack of sequential organization which one finds in these early stages of development, I do not know.

Another thing which has been impressive to me is the nature of the frequencies that one records from the central nervous system—the multiple or submultiple character of the rhythms one encounters, not only as a developmental feature of the maturing brain, but in the matured individual when the normal spontaneous rhythm breaks up and multiplies itself or divides in half. Just what relationship these changes in rhythm bear to actual behavior, again I do not know.

I have been particularly interested in trying to devise experiments to help get a better correlation between these superficially observed electrical phenomena of the cortex and the behavioral activities of the motor system. I find this very difficult to deal with in terms of sequential activities which are continually flowing along. It is very hard to devise a system which will do more than suggest possible correlates. On the other hand, there are phenomena we encounter both in the normal and the abnormal individual, which seem to bear some relationship to this serial order. I'd like to have Dr. Lashley comment if he will on whether the breaks in a sequence may not perhaps give us clues to some of the relationships. By this I refer to various types of blocking that one encounters, the blocking in the tapping of a motor rhythm, the blocking in the speech pattern of normal individuals and stutterers, the blocking in color naming, and a variety of others. Some of them (for example, in free associations) represent a longer time scale of blocking, but will do essentially the same thing. I have tried in some instances to correlate certain phenomena of the alpha rhythm with these patterns, such phenomena as the blocking of the alpha rhythm, or the out of phase character of the usually synchronized activity of the two cerebral hemispheres. I found, in some instances, very suggestive correlations, but I must confess that the proof that alpha blocking and the out of phaseness in the two hemispheres are related to blocking in behavior has not yet satisfied me.

DR. LASHLEY: I have been rather embarrassed by some of the flattering remarks made today. I think that here we must also consider background effects. Boring has pointed out that no progress is ever made until the time is ripe for that progress, and that the individual who makes the contribution is a very minor matter. If one doesn't, another will very shortly. What I have said today is really scarcely more than a compilation and summary of the thoughts of the people who have studied the aphasia problem. I have summarized again a series of problems which have confronted the students of aphasia for many years, to which Monakow and many others have contributed, Pick especially, and I have found their views helpful in considering such problems.

I have also been very much impressed with the similarity of the problems with which the behaviorist is confronted, and the problems with which the student of embryology must deal. The problem of form seems to me to be too similar in the two cases to be altogther due to different kinds of mechanisms.

There have been perhaps too many problems and questions raised here for me to attempt to answer them in detail. With regard to Professor Halstead's remark about the removal of my frontal lobes: In fantasy, I have thought perhaps that my most important contribution when I reach retirement age would be to have my frontal lobes removed and see what I could do without them. I have less confidence than Dr. Halstead that it would preclude the production of something of interest. We have little experimental evidence of intellectual defect from uncomplicated removal of the prefrontal lobes.

The conception of cerebral organization that I have sketched here is the result of repeated failures to confirm simpler hypotheses. Attempts to trace the course of conditioned reflex arcs or to discover the location of the traces of specific habits have been repeatedly unsuccessful. They have revealed areas specialized for different functions, but within those areas the parts have proved to be equivalent or equipotential for a great number of functions. Analysis of effective stimuli and of adaptive responses has shown that these are not dependent upon particular neurons but upon the spatial and temporal relations of excitation within shifting fields of nervous activity. Attempts to develop theories of neural integration in terms of chemical gradients or electrical potentials are difficult or impossible to relate to the established facts of axon conduction, however well they may seem to account for the facts of behavior.

I agree thoroughly with Dr. McCulloch that the transmission of excitation by the individual neuron is the basic principle of nervous organization. However, the nervous activity underlying any bit of behavior must involve so many neurons that the action of any one cell can have little influence upon the whole. I have come to feel that we must conceive of nervous activity in terms of the interplay of impulses in a network of millions of active cells.

In perception, in action, and in the preservation of memory traces there are systems or anatomic structures whose parts seem to be equipotential. The equipotential systems of which we have more definite knowledge all work by reduplication of elements or parts, of molecular or gross mechanical structure. The problem of how a reduplication of functional units might arise in the nervous system has seemed to me a basic one in neurology. The analogy with wave form and interference patterns is an attempt to meet that problem. In wave interference, patterns are reduplicated in different parts of the field, and I think it likely that nerve impulses, spreading at definite rates through a nerve net, may also produce reduplicated interference patterns. These would make possible the production of structural changes constituting memory traces at numerous points throughout the system. Such a concept is highly speculative, difficult to apply to concrete instances of behavior, but is as far as I have been able to go toward a solution of the problem of equipotentiality.

I mentioned briefly the problem of the dynamic level of nervous activity and the probable role of non-specific facilitation in the production of the shift from internal to overt speech or in determining the availability of memories. There are certainly other variables which affect the level of activity: changes in the chemical environment and the metabolic activity of the neurons. I believe that many of the symptoms following cerebral trauma or disease are due to such widespread chemical changes, resulting perhaps from vascular damage, rather than to actual destruction of nervous tissue. Aside from sensory defects, clinical symptoms more often suggest changes in dynamic level than the loss of specific integrations.

Dr. Köhler has raised the question of the stationary state of the world around us. I have tried to deal with this in terms of the constant space coordinates maintained by the postural system. The conceptual construct of the data obtained by scanning is closely integrated with this space system. I confess that I can form no clear picture of the actual physical mechanism.

Dr. McCulloch has given a beautiful illustration of the spatial determination of rhythmic activity by spread of excitation from a focal point of stimulation. I wish that such a mechanism were applicable to the problems of the syntax of action with which I have been dealing but I see no way in which such a simple mechanism can be used to account for the more complicated behavior. As for the mathematical representation of time as a fourth dimension: This may help to conceptualize motion, but the actual functions of the brain are carried out by a three-dimensional system. How is time or temporal order represented within the three dimensions? Mathematical symbolization of time as a fourth dimension does not suggest a clue.

Functional Differences between the Occipital and Temporal Lobes*

WITH SPECIAL REFERENCE TO THE INTERRELATIONS OF BEHAVIOR AND EXTRACEREBRAL MECHANISMS

HEINRICH KLÜVER
The Division of the Biological Sciences,
University of Chicago

I shall first briefly consider certain behavior alterations following bilateral removal of the occipital lobes in subhuman primates, more particularly, the changes observed in rhesus monkeys.

FUNCTIONS OF THE OCCIPITAL LOBES

I shall not describe the techniques I have developed for testing such animals (34–37, 40). Nor shall I bother you with the details of the experimental analysis. I shall simply point out that such animals in which the geniculostriate system has been eliminated have been studied for a long time before and after the operations. In some instances, thirty to forty-five trials per day have been given for more than 2000 days after the lobectomy. In studying the behavior of the bilateral occipital monkey I have been chiefly concerned with analyzing the responses of the dark-adapted animal to luminous stimuli differing in brightness, area, shape, color, and/or distance from the eye as well as the responses to intermittent light stimuli differing in light-dark ratios and flash frequencies per second. The results of this laborious analysis may be briefly summarized by saying that all or practically all differential reactions of the bilateral occipital monkey to visual stimuli can be understood by assuming the effectiveness of differences in the density of luminous flux at the eye or, expressed otherwise, differences in the quantity of light entering the eye (39, 41, 43). The eye of such

---

* Aided by the Dr. Wallace C. and Clara A. Abbott Memorial Fund of the University of Chicago and by the Commonwealth Fund.

an animal behaves like a photocell which merely registers and responds to fluctuations in intensity. It is worth noting that very small quantities of light are effective. In fact, the absolute threshold was found to lie between 4 and 20 micromillilamberts.

The question may be raised whether the absolute and relative thresholds remain constant with the passage of years after removal of the striate cortex. In this connection I should like to mention that I once gave Dr. Polyak the eyes of an adult rhesus monkey for an anatomical investigation of the retinae. He kindly undertook a thorough histological study of these retinae and reported that the left homonymous halves clearly showed a reduced affinity for the stains employed. At the time of this study Dr. Polyak was unaware of the fact that this particular monkey had undergone extirpation of the left occipital lobe four years previous to death (40). We must, therefore, consider the possibility that bilateral occipital lobectomy ultimately leads to very minute changes in the whole retina. In view of the possibility of retinal changes it will be a matter of considerable importance to determine in future studies whether the thresholds remain constant with the passage of time. On the basis of preliminary investigations I can merely say that very marked changes in absolute threshold values have not been found during a period of twelve years following removal of the striate cortex.

Our formulation that the bilateral occipital monkey is able to respond only to differences in the amounts of light falling on the retina implies that *all* visual functions are affected by eliminating the geniculostriate system. It does not imply that some visual functions are eliminated or seriously disturbed while others are left intact. Since visuospatial stimulus properties are no longer effective in determining visually guided behavior or, differently expressed, since visual space with its dimensions is completely or almost completely eliminated, it appears at first puzzling that the bilateral occipital monkey is capable of responding to differences in position even when the stimuli are simultaneously and continuously present. However, an experimental analysis of this differential behavior leads to the conclusion that the simultaneity of different visual stimuli in space is translated into a succession of different quantities of light received by the animal while occupying successively different positions in space. The fact that only differences in the quantity of light are effective has a positive side in the sense that the bilateral occipital monkey can make "generalizations" and perform responses which the normal monkey cannot or is not likely to perform. Except under special conditions, the normal monkey does not respond to the visual stimuli of the external world on the basis of differences in the density of luminous flux reaching the eye. For the normal animal the external world is structured in terms of objects with properties which stay put, that is, such properties as brightness, size, shape, and color.

Let us consider for a moment a normal monkey with an intact geniculostriate system. In numerous experimental situations involving the presentation of visual stimuli, I have found that for such a monkey the size of a visual object remains approximately constant despite marked changes in brightness, color, shape, position, and illumination (35). More generally speaking, size, brightness, color, shape, and certain other properties of visual objects are, within wide limits, not affected by numerous changes in the stimulus constellation. The intactness of the geniculostriate system, therefore, guarantees an approximate constancy of the objects and their interrelations in the external visual environment.

To illustrate what it means that certain properties of the objects of the external environment remain stable, let me refer to a squirrel monkey (with about 22 grams of brain) which, in some experiments about 14 years ago, faced the external world in my laboratory and discovered that, in order to eat, it must learn to choose one of two objects presented. It must choose a yellow object in the presence of a green one, it must choose green in the presence of red, red in the presence of blue, and blue in the presence of yellow. When the yellow-green, green-red, red-blue, and blue-yellow color pairs were presented in chance order, the monkey made the correct choices without great difficulty and consequently was able to eat and live. How is such a performance possible? There is strong evidence that the squirrel monkey has, as we say, color vision. And having color vision implies the approximate constancy of "hues" in the presence of variations in area, shape, brightness, distance, illumination, etc. If a certain property of an object, such as hue, stays put it may serve as a point of departure for diverse behavioral reactions, including "thinking." The squirrel monkey is, in effect, able to say: If *a*, then *b;* if *c*, then not *a;* etc. I have shown that destruction of the striate cortex in rhesus monkeys produces loss of color vision. In determining the effectiveness of different portions of the spectrum I have obtained relative effectiveness curves similar to the scotopic luminosity curve of the human eye, but the approximate constancy of hue in the presence of numerous variations has disappeared in such animals.

Physiologists have rightly insisted that the organism is able to fall back on numerous mechanisms which guarantee an approximate constancy of the internal environment. Such mechanisms serve, for instance, to maintain what has been called the "isochemistry" of the blood. Analysis of the functions of the occipital lobes has led me to conclude that the intactness of the geniculostriate system enables the organism to fall back on mechanisms which guarantee an approximate constancy of the external environment. I submit that the Harvard emphasis on a homeostasis in the *milieu interne* should be counterbalanced by a Chicago emphasis on a homeostasis in the *milieu externe*. If, in the absence of the geniculostriate system, the animal is forced to fall back on functions mediated by the retina and certain subcortical structures of the "visual sector" of the central nervous system, it finds itself at the mercy of ever-changing energy fluctuations in the external environment because of a practically complete loss of visual isofunctions. Since the remarkable degree of "freedom" from fluctuations in the visual milieu enjoyed by the normal animal is linked up with the existence of the geniculostriate system, it is not surprising that an elimination of this system has particularly serious consequences for "optoid" animals, such as primates. In primates, the *systema nervorum pro mundo* deals with a "world" built up chiefly via the visual route. In closing my discussion of the functions of the occipital lobes I wish to say that I have aimed at formulating broad generalizations. I believe, however, that these generalizations are warranted by experimental results.

FUNCTIONS OF THE TEMPORAL LOBES

Let us now turn to some behavior changes which probably represent the most striking behavior changes ever produced by a brain operation in animals. These are the behavior changes which appear in sexually mature rhesus monkeys after bilateral removal of the temporal lobes, including most of the uncus and hippocampus. In considering the relation between animal and external environment, the somewhat oversimplified and paradoxical statement may be made that removal of the occipital lobes only changes the environment while removal of the temporal lobes only changes the animal. About twenty-five years ago, Spitzer (76), in discussing the anatomy and physiology of the vestibular system, made the distinction between an idiotropic nervous system concerned with interrelating impulses within the organism—thus achieving a unification for the sake of self-preservation—and an oikotropic nervous system concerned with relating the organism as a whole to stimuli of the external environment. Spitzer insisted that oikotropic zones are more laterally located than idiotropic zones. For example, the cochlear nuclei with oikotropic functions are more laterally situated than the idiotropic vestibular nuclei. In Spitzer's terms, therefore, it might be said that the occipital lobes are associated with oikotropic functions whereas the temporal lobes are associated with idiotropic functions.

Leaving aside formulations of such general nature, I shall now turn to the behavior alterations observed in the bilateral temporal monkey. The various changes following bilateral temporal lobectomy in macaques have been studied in collaboration with Dr. P. C. Bucy. Since the results of these experiments have been previously published (49–52) I shall confine myself to a brief description of the outstanding symptoms.

(1) We observe, first of all, forms of behavior which appear to indicate a "psychic blindness" or visual agnosia in the sense that the ability to recognize and detect the meaning of objects on the basis of visual criteria alone seems to be lost although the animal exhibits no or at least no gross defects in the ability to discriminate visually. There is a strong tendency to approach every animate and inanimate object without hesitation, even objects which previous to the operation called forth extreme excitement, avoidance reactions, and other forms of emotional response. When various edible and inedible objects are presented at the same time, the caged-in monkey, even when hungry, picks up all objects indiscriminately. After an oral examination the inedible objects, such as a steel nut, a piece of glass, a wad of tin foil, feces, a live mouse, or a piece of cloth, are discarded while the edible objects are consumed. The animal may examine the tongue of a hissing snake, the eyebrows of a human being, a screw driver, or the outlines of a figure drawn on paper as eagerly and as carefully as a piece of food.

(2) There are strong oral tendencies in the sense that the monkey insists on examining all objects by mouth. Such an oral examination consists in putting the object into the mouth, licking, biting gently, chewing, touching with the lips, and "smelling" by holding the object before the nostrils. We also observe oral tendencies when the animal, free in a room, tends to touch various objects directly by mouth instead of using its hands. The monkey even tends to examine orally objects which have previously acquired "meaning" through training, such as a cardboard circle signaling the presence of food in a certain location.

(3) There is an excessive tendency to attend and react to every visual stimulus. The monkey behaves as if forced, under the influence of some "irresistible" impulse, to contact all objects in sight. In other words, we find, to use Wernicke's terms, a "hypermetamorphosis" and a "hypermetamorphic impulse to action." Seeing an object and

performing the necessary motor reactions for touching it seem to be a continuous process.

(4) There are profound changes in emotional behavior, and there may even be a complete loss of emotional responses in the sense that such forms of motor and vocal behavior as are associated with anger and fear in the rhesus monkey are not exhibited. The very fact that the animal approaches and touches every object, whether it be a stranger, a cat, a dog, a snake, or an inanimate object, must be considered a striking deviation from normal behavior. The monkey even approaches and tries to touch another monkey which has just attacked and injured it. All expressions of emotions, such as "chattering" and various facial expressions, may be completely lost.

(5) There is a striking increase in the amount and diversity of sexual behavior. The animal appears hypersexed not only when caged with other animals, but also when alone. It exhibits forms of autosexual, heterosexual, and homosexual behavior rarely or never seen in normal monkeys. One animal, an adult Guinea baboon, had to be killed because of the serious nature of self-inflicted injuries brought about by again and again biting its penis and legs. Even a pseudohermaphrodite exhibited an intensification of sexual responses. It is to be noted that our observations on changes in sexual activity are chiefly based on males.

(6) There occurs a remarkable change in dietary habits manifesting itself in the tendency to consume large quantities of ham, bacon, boiled tongue, ground beef, broiled lamb chops, smoked whitefish, and other kinds of meat offered.

This, in brief, is the complex picture of behavior alterations following bilateral temporal lobectomy. Let us now return for a moment to the heroic age of cerebral physiology, for instance, to the year 1888, that is, to a period in which the "isms," "schools," and "57 varieties of psychology" were still a matter of the future, and let us see how it is possible to describe the behavior of a bilateral temporal monkey in plain English. After a rather thorough study of the behavior changes following removal of the temporal lobes I could not reconcile myself to the fact that such changes had never been reported by previous investigators although temporal lobes had been removed from various animals, including monkeys, in numerous physiological laboratories in Europe as well as in this country. I searched the world literature again and was rewarded by finding that Sanger Brown (who later became the first president of the Chicago Neurological Society) and E. A. Schäfer, professor of physiology in University College, London, had seen, more than half a century ago, two monkeys exhibiting essentially the same symptoms I have described. These two monkeys are monkeys VI and XII referred to in the paper, "An Investigation into the Functions of the Occipital and Temporal Lobes of the Monkey's Brain" (8). And this is the way Sanger Brown and Schäfer described in 1888 the behavior alterations following removal of both temporal lobes from their monkey VI ("a fine, large, active" male rhesus monkey):

> A remarkable change is . . . manifested in the disposition of the Monkey. Prior to the operations he was very wild and even fierce, assaulting any person who teased or tried to handle him. Now he voluntarily approaches all persons indifferently, allows himself to be handled, or even to be teased or slapped, without making any attempt at retaliation or endeavouring to escape. His memory and intelligence seem deficient. He gives evidence of hearing, seeing, and of the possession of his senses generally, but it is clear that he no longer clearly understands the meaning of the sounds, sights, and other impressions that reach him. Every object with which he comes in contact, even those with which he was previously most familiar, appears strange and is investigated with curiosity. Everything he endeavours to feel, taste, and smell, and to carefully examine from every point of view. This is the case not only with inanimate objects, but also with persons and with his fellow Monkeys. And even after having examined an object in this way with the utmost care and deliberation, he will, on again coming across the same object accidentally even a few minutes afterwards, go through exactly the same process, as if he had entirely forgotten his previous experiments. His food is devoured greedily, the head being dipped into the dish, instead of the food being conveyed to the mouth by the hands in the way usual with Monkeys.

It is evident that the observations of Sanger Brown and Schäfer agree fully with ours, particularly with reference to the picture of psychic blindness, the oral tendencies, the "hypermetamorphosis," and the striking changes in emotional behavior. Although there is a striking agreement as regards the description of the symptoms, I cannot agree with the interpretation of the authors that these symptoms are indicative of a condition resembling "general idiocy." However, anyone who has ever seen the truly remarkable behavior changes following bilateral temporal lobectomy will not be surprised to learn that Sanger Brown and Schäfer felt called upon to demonstrate their two monkeys before various scientific societies (8, 69). In fact, these "two animals were exhibited to the Neurological Society, and a Committee of that Society was appointed to examine them" (69). As regards the lesions that produced these changes in behavior, David Ferrier expressed the opinion that Schäfer's published diagrams of the brains of these monkeys "must be regarded more as a sort of pictorial representation of his own idea, or wish, as to the extent of the lesions, rather than of the reality" (22). Schäfer, who had claimed, for instance, that he removed "the whole of the temporosphenoidal lobe, including the hippocampus," on both sides, was fortunately able to publish photographs of these brains. It is evident from these photographs (70) that the bilateral lesions, for example, in monkey VI, were comparable in extent to the lesions produced in our experimental animals.

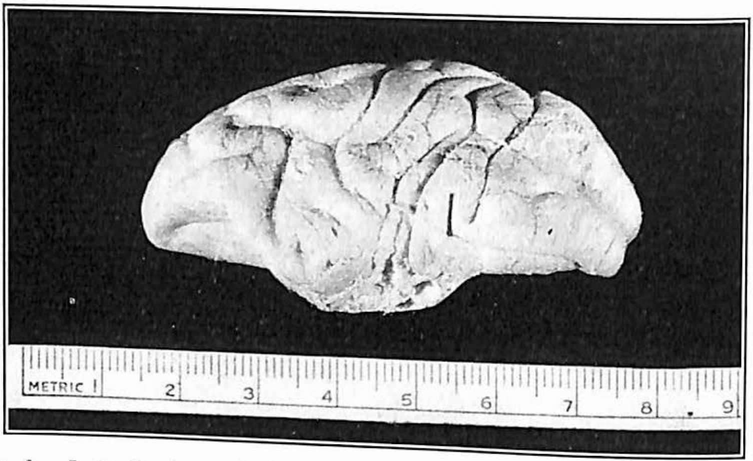


FIGURE 1. Lateral view of the left hemisphere of brain of Case 1 (rhesus monkey).

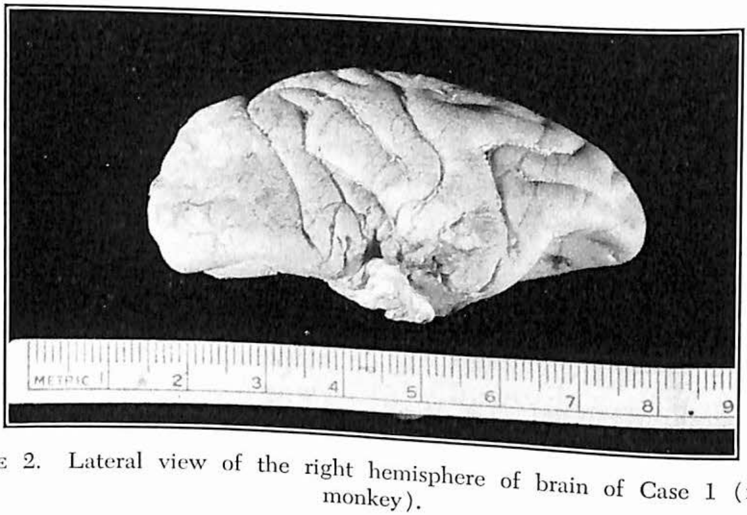


FIGURE 2. Lateral view of the right hemisphere of brain of Case 1 (rhesus monkey).

For a better understanding of the behavior alterations in our monkeys it is necessary to have detailed information not only on the extent of the lesions, but also on the tracts and nuclear structures that have subsequently undergone degeneration. I shall briefly indicate, therefore, the anatomical changes secondary to bilateral temporal lobectomy by referring to the results obtained in studying the brain of the first animal in our experimental series [Case 1 in (52)]. This animal was killed 22 months after removal of the temporal lobes. Figures 1 and 2 show photographs of the brain taken from the left and the right side. Since the lesions were almost symmetrical, photomicrographs of representative sections from only one of the hemispheres are shown in Figures 3 to 9. The anatomical findings may be summarized as follows (9):

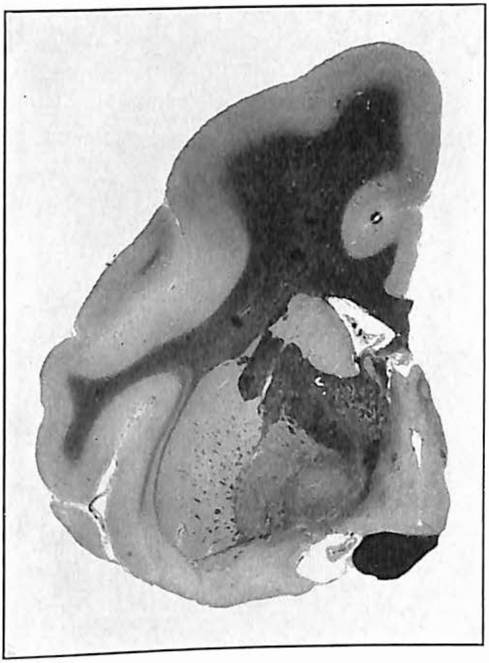


FIGURE 3. Coronal section through left hemisphere. Weil stain.

(1) Both temporal lobes are removed. The line of extirpation extends posteriorly along the lower border of the Sylvian fissure to a point just posterior to the lower end of the fissure of Rolando (Figure 3). Here it turns ventrally to cut across the first and second temporal convolutions. As regards the rhinencephalon, only a fragment

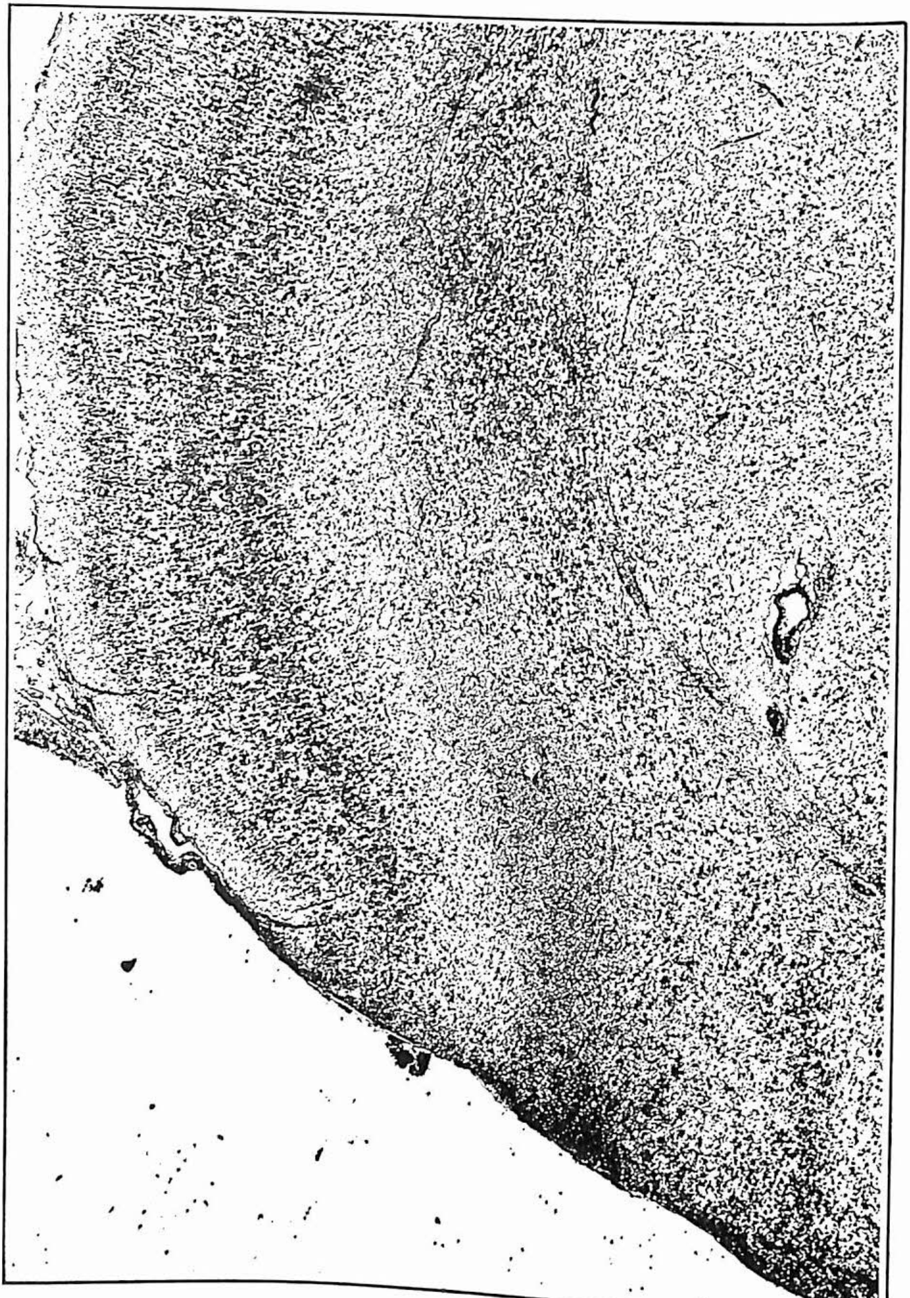


FIGURE 4. Photomicrograph from a coronal section through left hemisphere. Cresyl violet stain. ×25.

of the right amygdala and the most posterior part of the hippocampal formation are preserved.

(2) The frontotemporal fibers forming the uncinate fasciculus (Figure 3) and a temporo-olfactory bundle are degenerated. The latter, after passing medially beneath the putamen and the caudate nucleus, crosses in the rostrum of the corpus callosum to the olfactory trigone of the opposite hemisphere. The degenerated tracts passing upward from the plane of amputation in the capsula extrema are clearly outlined by gliosis in Figure 4.

(3) There is a degeneration of fibers passing into the remaining parts of the first (Figure 6) and second temporal gyri. Fibers passing from the temporal lobe upward into the posterior frontal and parietal areas and backward into the occipital region are also degenerated although to a less marked degree.

(4) There are two large groups of degenerated fibers, one of which passes to the posterior part of the cingulum and the other through the posterior part of the corpus callosum to the opposite hemisphere. Figure 6 shows a bundle of demyelinated fibers passing dorsomedially from the corona radiata over the lateral ventricle into the cingulum. It also shows a bundle of demyelinated fibers in the ventral one-third of the corpus callosum.

(5) The anterior commissure is completely degenerated except for an extremely small ventral bundle (Figure 5).

(6) A small ventral part of the corticotectal system passing from Brodmann's field 19 to the superior colliculus is degenerated. The small region of gliosis visible in Figure 7 indicates the position of the degenerated corticotectal fibers.

(7) A rather large tract terminating in the substantia nigra is degenerated. In Figure 9 this tract can be seen passing through the damaged ventral portion of the putamen and curving over the lateral geniculate body.

(8) A large bundle of fibers known as the temporopulvinar or Arnold's bundle is degenerated. Figure 7 shows the marked gliosis of this bundle in the ventral part of the inferior nucleus of the pulvinar.

(9) The hippocampal formation is extensively removed. Only small remnants of the posterior part of this formation are preserved on the left (Figure 7) and the right (Figure 8). As a result the fornix is extensively degenerated (Figures 5, 6, and 8). Figure 5 shows the demyelinization of the lateral three-fifths of the body of the fornix just at the beginning of the columns.

(10) The portion of the tail of the caudate nucleus which lies in the temporal lobe is extirpated. There is a partial degeneration of

the stria terminalis (Figure 8). In addition, a small part of the posteroventral putamen is damaged.

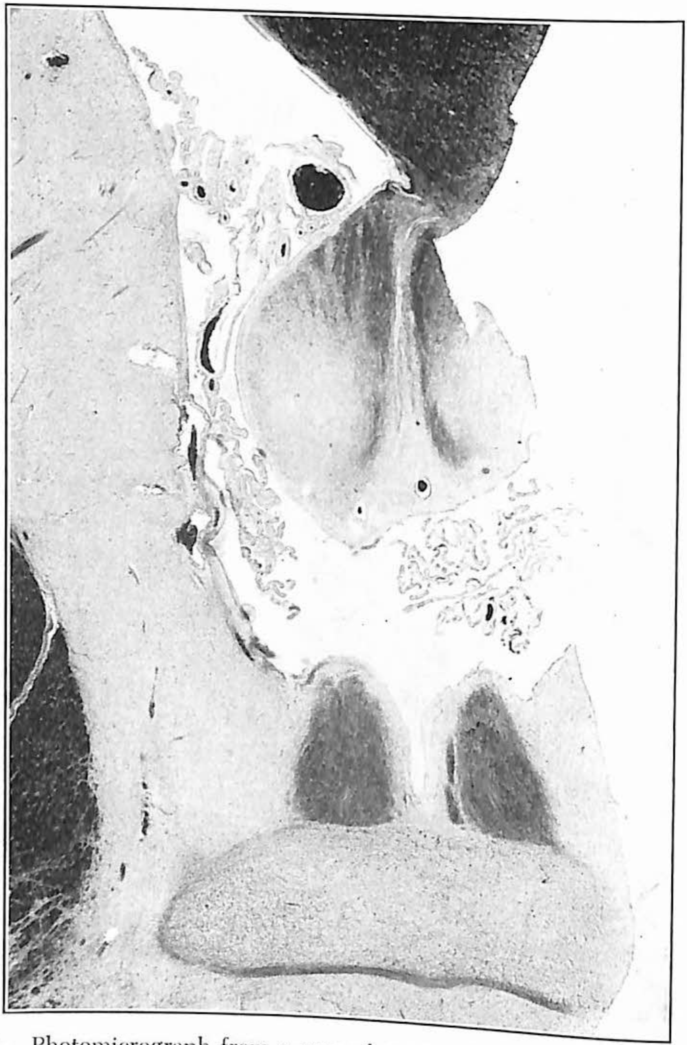


FIGURE 5. Photomicrograph from a coronal section through region of the anterior commissure. Weil stain. ×15.

(11) In the thalamus, cellular degeneration is confined to a small part of the inferior nucleus of the pulvinar and to the posterior tips of the medial and lateral nuclei of the pulvinar (Figure 8).

(12) In the visual system, a small portion of the most ventral fibers of the visual radiation on the left side is interrupted. Consequently,

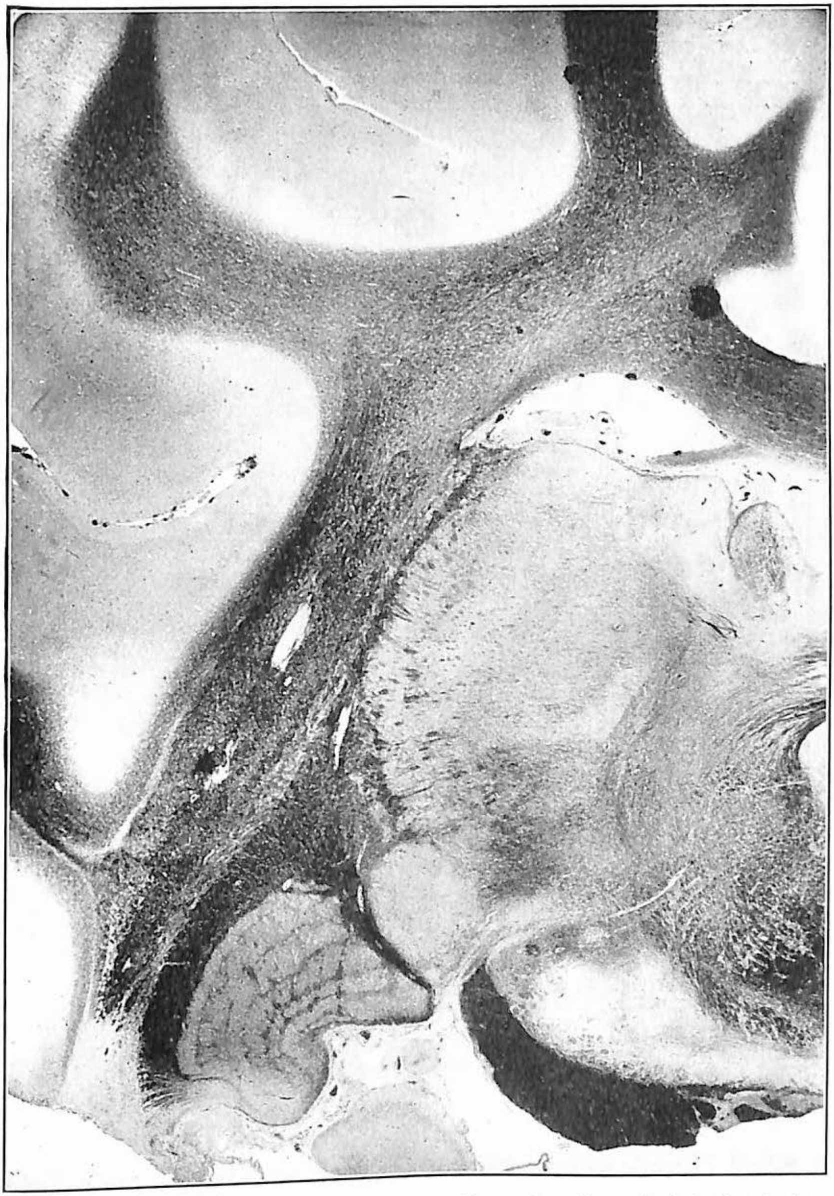


FIGURE 6. Photomicrograph from a coronal section through left hemisphere. Weil stain. ×7.

there is a small area of retrograde degeneration in the ventrolateral part of the lateral geniculate body (Figures 6 and 9). Destruction of the ventral fibers of the visual radiation on the right side is somewhat more extensive, resulting in a correspondingly larger area of retrograde degeneration in the lateral geniculate body. In the auditory system, retrograde cellular degeneration is present in the posterior tips of the medial geniculate bodies.

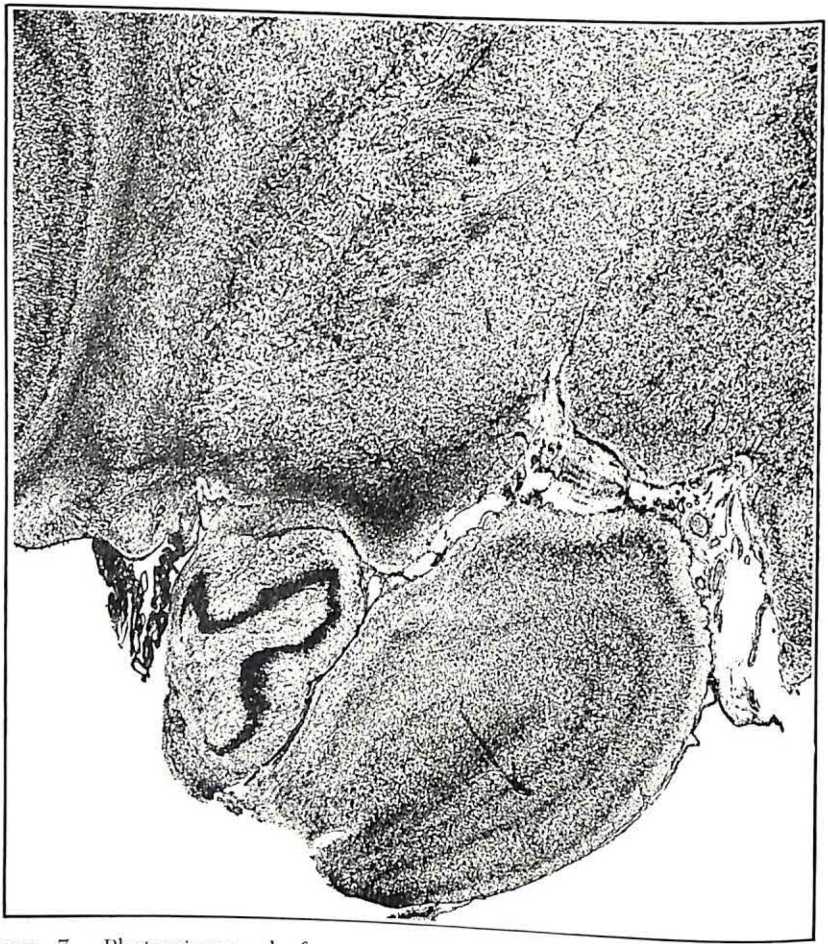


FIGURE 7. Photomicrograph from a coronal section through left hemisphere. Cresyl violet stain. ×11.

It should be remarked that the anatomical study of additional brains in our series has not disclosed findings essentially different from those reported here.

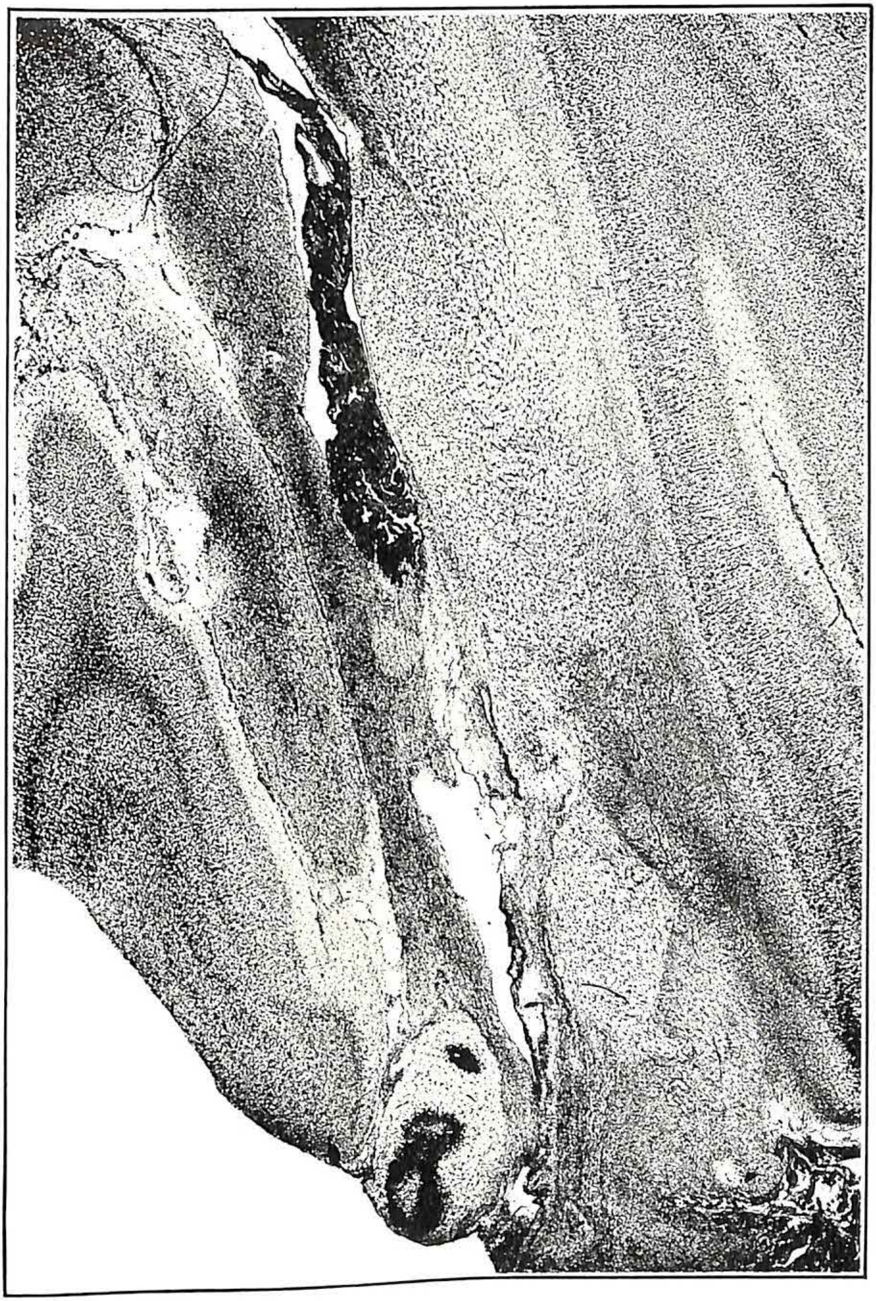


Figure 8. Photomicrograph from a coronal section through right hemisphere. Cresyl violet stain. ×15.

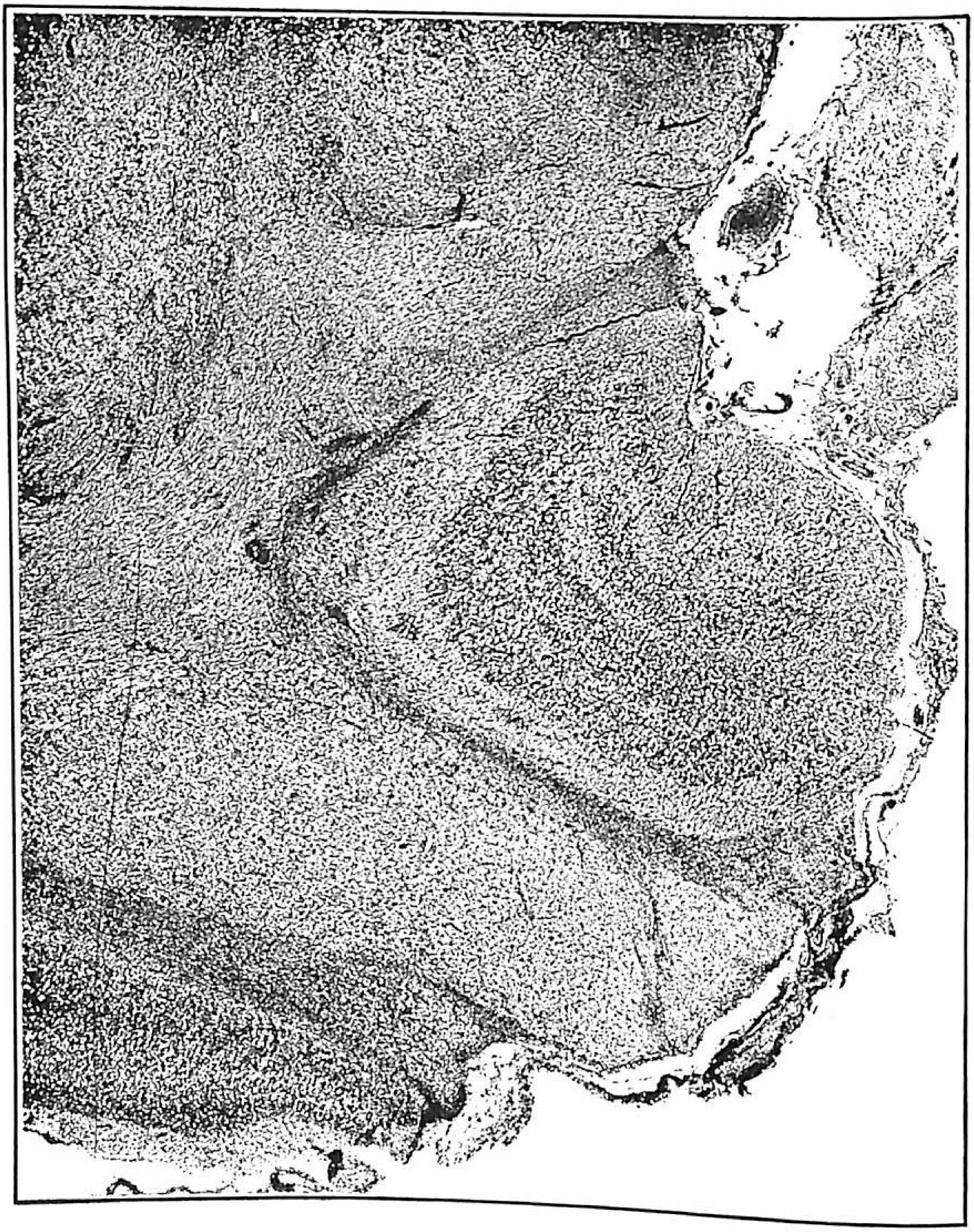


FIGURE 9. Photomicrograph from a coronal section through left hemisphere. Cresyl violet stain. ×13.

Our brief summary raises the question as to how the complex picture of behavior alterations is related to the equally complex picture of anatomical alterations in the brain. It is not possible at this point to go into further details of our experimental analysis of the behavior changes. Nor is it possible to discuss the interesting changes in symptomatology with the passage of time or the role of such factors as age, sex, and species. It seems tempting to reduce the complexity on the behavior side by considering the polysymptomatic picture as an expression of a disturbance in some one fundamental mechanism. For instance, it may be argued that the factor underlying the variety of behavioral manifestations is represented by an excessively strong tendency to approach and touch every object in sight, no matter what its character. The tendency to approach, attack, touch, or imbibe objects of the external world has frequently been contrasted by students of animal behavior with the tendency to retreat, avoidance or flight. It is of interest that von Monakow (56) insisted on a similar distinction by introducing *klisis* and *ekklisis* as fundamental tendencies operative in behavior. To refer all symptoms following removal of the temporal lobes to a loss of von Monakow's *ekklisis*, however, is obviously an oversimplification. The experimental data available at the present time rather suggest that the symptoms involve heterogeneous mechanisms which happen to be thrown into action at the same time when certain anatomical structures and their connections are damaged or eliminated.

In considering the complex picture of anatomical changes it is difficult to say how particular changes, such as the degeneration of temporo-olfactory fibers or of Arnold's bundle, are related to particular behavior alterations. The fact that the temporal neocortex, with exception of the auditory koniocortex, is the only portion of the cerebral cortex which receives few, if any, afferent impulses from the thalamus (80–82) is no doubt functionally significant, but it is of little help in trying to understand why an elimination of the whole temporal lobe should produce the observed behavior alterations. By studying the effects of bilateral removal of portions of the temporal lobes and the effects of variously situated lesions in other parts of the cortex, Dr. Bucy and I have attempted to determine the minimal lesion necessary for producing the behavior changes. Although these experiments have not led to definite conclusions, they strongly suggest that the hippocampal formation and its connections play an important role in the production of the symptoms. It is pertinent to mention at this point the interesting hypothesis advanced by Papez (61) "that the hippocampus participates in some important way in the central production of the emotive process," more specifically, "that the hypothalamus, the anterior thalamic nuclei, the gyrus cinguli, the hippocampus and their interconnections constitute a harmonious mechanism which may elaborate the functions of central emotion, as well as participate in emotional expression." Papez finds no evidence to support the view that these anatomical structures mediate olfactory function, and Brodal (6), in a recent anatomical study, reaches essentially the same conclusion in discussing the relation of the hippocampus to the sense of smell. Furthermore, Kleist (32) independently advanced a hypothesis similar to that of Papez on the basis of clinical observations, except that he included the frontal *Orbitalhirn* in his anatomical circuit. Guided by the hypotheses formulated by Papez and Kleist, future experimenters may profitably direct their work toward determining whether interruptions of these anatomical circuits at various points by appropriate lesions are equivalent in producing the same picture of behavior alterations. At present there is no evidence that the entire picture of behavior changes can be produced except by bilateral removal of both temporal lobes, including the nuclei of the amygdala, the uncus, and almost the entire hippocampus. However, it has been reported that some of the symptoms, at least in an attenuated form, may appear after removing the rostral cingular gyrus (71, 84). Various investigations have thrown new light on the functions mediated by the cingular gyrus as well as by other structures involved in Papez' anatomical circuit (1–5, 59, 60, 64, 68, 72, 73, 83, 88). We still have no detailed information on the behavioral effects produced in monkeys by an isolated removal of the entire hippocampus on both sides. Our own experiments merely indicate that partial removal of this region leads to a transitory appearance of some of the symptoms.

The question may be raised whether the whole picture of behavior changes following extirpation of the temporal lobes can be adequately understood simply by considering the destruction of certain fiber tracts and nuclear structures in the brain. For example, the tendency to consume animal foods is probably not the result of an impairment in gustatory sensitivity, but involves entirely different physiological mechanisms. In experiments performed by Chaikoff and co-workers (10), the right temporal lobe of dogs was retracted after making cranial and dural openings, and the hypophysis and the base of the brain were exposed to view for one minute or less. This procedure led to a marked increase in sensitivity to insulin. In fact, in one of the dogs a 46 per cent reduction in blood sugar could be produced even three months after the operation by injecting $\frac{1}{16}$ unit of insulin per kilogram of body weight. These findings suggest the possibility that the extensive removal and subsequent degeneration of tracts and structures in our monkeys, particularly of the rhinencephalon, have affected hypothalamico-hypophyseal interrelations with resulting disturbances in hypophyseal function. Disturbances in carbohydrate metabolism and in other functions referable to the hypophysis or to hypothalamico-hypophyseal mechanisms may thus produce some of the behavior alterations seen in our monkeys. Eating meat may simply represent one of the behavior regulations concerned with maintaining a constant internal environment. The frequent occurrence and diversity of such behavior regulations has been demonstrated by Richter (65) in numerous experiments on the self-selection of diets. In a somewhat similar manner, the striking hypersexed behavior may be related to changes in the hormone production of the anterior pituitary. In future studies, the daily urinary excretion of androgens and estrogens should be of interest. It is probably significant that one of the females in our series exhibited irregularities in menstruation from the time the temporal lobes were removed until the time of death. Her prefrontal lobes had been removed in previous operations. When the animal died six years and nine months after the extirpation of the temporal lobes the necropsy revealed old and new hemorrhages and extensive adhesions in the abdominal cavity. Microscopical study of the uterus, Fallopian tubes, and ovaries disclosed an endometriosis. This is probably the first case of an endometriosis in a laboratory monkey. At a time when thousands of human patients are about to be lobectomized, lobotomized, and gyrectomized, the possibility should be kept in mind that surgical procedures on the brain may initiate processes which lead to extracerebral pathology one-fourth of a lifetime later, particularly if such procedures have affected conditions in or projections to the hypothalamic area (55, 57, 85). At any rate, there is abundant evidence from the work of Ranson and collaborators (7, 16–20, 54) that direct injury of the hypothalamic region in female guinea pigs may produce disturbances in reproductive functions and sexual activity as well as changes in the uterus and ovaries. By way of interpreting such facts, these investigators have found it necessary to consider not only the destruction of neural structures, but also the effects of "ovarian or anterior pituitary hormonal imbalances" associated with hypothalamic injuries. In the opinion of Dey (17), the final explanation for the hypophyseal dysfunction following hypothalamic lesions must await further experimentation.

BEHAVIOR WITH REFERENCE TO STRUCTURAL AND STIMULUS FACTORS

Numerous studies concerned with the relation between cerebral mechanisms and behavior mechanisms confine themselves to relating the occurrence or non-occurrence of behavior changes to the presence or absence of lesions in the brain. Our analysis, particularly of the behavior changes following removal of the temporal lobes, has thrown doubt on the assumption that behavioral manifestations can always be fully understood by merely considering the destruction of structures in the brain. In trying to interpret our experimental findings we have found it necessary, for example, to discuss the role of extracerebral mechanisms. Since in many attempts at correlating brain mechanisms and behavior mechanisms "a parallelism is still credited," as Wertham (87) has expressed it, "which is not only psycho-physical, but also psycho-histological," it becomes imperative to discuss behavior in relation to cerebral and extracerebral mechanisms from a broader point of view.

Specific correlations often seem warranted in view of the fact that particular lesions or particular structures are found to be associated with certain forms of behavior observed when the organism is exposed to particular stimuli, whether they be environmental stimuli, chemical agents, or other stimuli. However, a more detailed experimental analysis often provides little or no support for such specific correlations. In the following I shall try to show why past attempts to establish such specific correlations have frequently met with failure. With this aim in view I shall first refer to certain experimental studies concerned with an analysis of the mechanisms involved in the "experimental catatonia" produced by bulbocapnine.

In presenting a summary of previous work, Ferraro and Barrera (21) pointed out in 1932 that "most of the authors have concluded that the neocortex appears to be essential to the manifestation of typical catalepsy. . . ." In fact, they found that previous investigators had particularly emphasized the importance of the motor cortex in the development of the symptoms. In their own work, Ferraro and Barrera attempted to find "some central or peripheral nervous system lesion which might substantially influence or abolish the action of the drug. . . ." They performed sixteen different operations on monkeys and cats, including splittings of the mesencephalon, cutting of the dorsal and ventral roots, removal of the abdominal sympathetic chain, and bilateral stellate ganglionectomy. None of these lesions prevented the occurrence of cataleptic manifestations under the action of bulbocapnine. They were thus led to the generalization that the action of bulbocapnine "involves contemporaneously the central nervous structures, the peripheral nervous structures, and possibly, the muscular structures themselves." As regards the muscular structures, they suggested an involvement of the sarcoplasm of the muscle fibers. It seems, indeed, a far cry from the assertion that the neocortex or, perhaps, only the motor cortex is an essential factor in the production of catalepsy to the assertion that the whole nervous system and possibly the sarcoplasm of the muscle fibers are involved. Ferraro and Barrera finally concluded that "any theory based upon a localized center for the production of catalepsy by bulbocapnine will be erroneous in its deductions." I am not concerned here with evaluating the various results obtained in studying catalepsy in animals, but merely with demonstrating why attempts at correlating particular behavior manifestations with specific structures may fail. They often fail because more extensive experimentation does not provide evidence for a specificity on the *structural* side.

Let us now turn to the stimulus side, that is, to the chemical agent producing the cataleptic manifestations. In the course of his studies on the action of bulbocapnine, de Jong discovered that other substances, including epinephrine and acetylcholine (13, 58), also produce catalepsy in animals. He concluded that there may be a chemical "catatonizing nucleus." In the meantime, further experimentation has led to the discovery of even more chemical agents capable of producing cataleptic manifestations, but it has not disclosed the existence of a chemical "catatonizing nucleus" (14). It seems, then, that long-continued research may also fail in defining specificities on the *stimulus* side, that is, in locating the particular stimulus property or set of properties effective in producing a given form of behavior.

The fact that heterogeneous stimuli, such as, for instance, different chemical agents, may produce the same or similar effects raises questions which are of fundamental importance for a scientific analysis of behavior. It is true that various biochemical and pharmacological investigations have established certain correlations between physiological activity and the chemical constitution of inorganic and organic substances (23). It can be shown, for example, that certain similarities in the physiological effects of epinephrine and related compounds are correlated with certain similarities in the chemical constitution of these compounds (26). It can also be shown that similar behavioral effects may be produced by the action of chemically similar substances. In studying the effects of mescaline (3, 4, 5-trimethoxyphenethylamine) in monkeys, I observed that this drug produces a characteristic "oral syndrome" consisting of movements of the lips, tongue, and jaws and involving peculiar licking and chewing movements. I found that the same or similar symptoms are also produced by 3, 4-dimethoxyphenethylamine, 3, 4-diethoxyphenethylamine, 3, 4-methylenedioxyphenethylamine, α-methylphenethylamine, phenethylamine, tyramine, ephedrine, cobefrine, benzylamine, and aniline. It seems, therefore, that the production of similar "oral syndromes" can be correlated with certain similarities in the chemical structure of the compounds. However, further research revealed that similar syndromes can also be produced by pentobarbital sodium and apomorphine.

The fact that chemically dissimilar substances may produce typical mescaline effects can be even more strikingly demonstrated in the psychic sphere. It is chiefly because of its remarkable psychological effects that mescaline has become a research tool in the hands of psychiatrists. It produces not only certain types of hallucinations, but also characteristic changes in different sense fields and certain alterations in thinking and affectivity (33, 42). There is no definite evidence from our own observations or the literature that the train of phenomena induced by mescaline can be duplicated by administering substances which are chemically closely related to this drug. It is of great interest that such a duplication in psychological effects can apparently be obtained by employing one of the ergot derivatives, namely, *d*-lysergic acid diethylamide. Even the oral administration of 30 micrograms of this substance produces such strikingly similar symptoms that the subject, as Stoll (77) points out, uses "literally the same figures of speech and expressions" in describing his experiences as a mescalinized person.

While a psychologist may want to know why substances as different as mescaline and *d*-lysergic acid diethylamide produce the same complex picture of psychic alterations, a pharmacologist may enquire why harmine and quinine produce the same syndrome of physiological effects. Gunn (25), who discovered that the harmine and quinine groups of alkaloids act similarly on protozoa and on such different tissues as heart, uterus, voluntary muscle, and central nervous system, raised the question how "a common group of effects" can be produced "by two molecules which have (apparently) nothing in common." Similar questions arise when a neuroanatomist finds that traumatic, circulatory, toxic, and infectious processes produce the same kind of lesion. Spielmeyer (75) was, therefore, greatly concerned with determining why "fundamentally different diseases" and "very diverse noxious factors" may produce the same histological changes in the central nervous system.

The various observations cited here from different fields furnish examples of what I have called an "equivalence of stimuli" (35, 38). Since heterogeneous stimuli, such as diverse chemical substances, disease processes, or sensory stimuli, often produce the same series of effects, it becomes obviously necessary to account for such an equivalence of stimuli by specifying the set of properties or the common factor involved in producing the same effect. Unfortunately, the properties apt to account for an equivalence of heterogeneous stimuli have seldom been adequately defined. Although the specification of the factors underlying particular forms of equivalence is of paramount importance for the study of behavior, most attempts along such lines have either failed or merely succeeded in characterizing such factors in a very general fashion. As previously mentioned, all attempts to find a chemical "catatonizing nucleus" to account for the equivalence of diverse chemical agents capable of producing experimental catalepsy in animals have met with failure. To account for the equivalence of harmine and quinine in producing the same syndrome of physiological effects, Gunn (25) postulated "a common chemical factor in the tissues acted upon," although he considered the possibility of a "concealed" chemical similarity between the two molecules. To account for the equivalence of diverse noxious factors in producing the same damage in the nervous system, Spielmeyer (75) had recourse to common pathophysiological mechanisms. It seems, then, that there is no lack of general theories or vague hypotheses purporting to account for particular forms of equivalence. As regards the equivalence of different chemical agents, the effects observed are often of such a nature that the investigator abandons all hope of explaining the results on a chemical basis. For example, Degkwitz (12) concluded that any explanation ignoring physical properties is totally inadequate when confronted with the fact that about 480 molecules of irradiated ergosterol for every cell of the body cure rickets in a rat within 8 days. Fränkel (23) insisted that any theory taking into consideration only one set of factors in explaining physiological effects, whether they be physical or chemical factors, must be "wrecked by the multiplicity of interrelations" among different chemical substances and different tissues.

Facts of equivalence come to light not only when the organism is studied along pathological, physiological, biochemical, and anatomical lines, but also when behavioral and psychological reactions are analyzed. Not only do heterogeneous stimuli often produce the same behavioral effect, but the same stimulus may be dealt with by diverse actions and reactions. We find such facts of equivalence on all levels of behavior, ranging from reflexes to the most complex reactions of normal and abnormal personality. In attempting to specify the factors responsible for particular forms of equivalence, I have usually only succeeded in defining such factors negatively. In other words, I have often been able to eliminate one factor after another by demonstrating that various common properties or sets of properties found in a given group of equivalent stimuli play no role in bringing about the same behavior (35).

How extremely difficult it may be to cope theoretically with facts of equivalence not only in the field of behavior, but also in other fields, may be illustrated by Speransky's finding (74) that "rabies, a sore on the *tuber cinereum* produced by a glass sphere, irritation of any branch of the *n. trigeminus* by croton oil, injection of formalin into the pulp cavity of a tooth or of bile into the upper cervical ganglion, introduction of foreign protein or various vaccines into the blood, poisoning by salts of the heavy metals, e.g., corrosive sublimate, etc.—all these are capable of producing, in a number of organs, changes that are *absolutely constant and so much alike that it is impossible to distinguish them from one another*." Assuming that the facts are as reported by Speransky, we have here an equivalence of such diverse stimuli that any attempt to seek a common factor in the properties of the stimuli employed must seem futile. It appears obvious that the effects observed are not related to a property common to such different agents as a glass sphere, corrosive sublimate, and rabies virus. Speransky is undoubtedly right in concluding that we must look for "constant features which do not depend on the properties of the irritating agent. . . ." This means, then, that there are forms of equivalence which cannot be satisfactorily understood by reference to the properties of the stimuli employed. If practically any form of stimulation or irritation produces the same effect, the constant factors must be located in the reaction systems, processes, and conditions within the organism.

Our discussion so far has dealt with some of the complex problems that may arise in attempting to relate brain mechanisms and behavior mechanisms. We have shown that a detailed experimental analysis of these interrelations may force us to consider, on the one hand, not only cerebral mechanisms, but also such extracerebral factors as processes and conditions in other organs and systems and, on the other hand, not only the various properties of the stimuli and agents which happen to be employed in influencing behavior, but also the nature of the behaviorally effective properties. It is clear that we must rely on future experimentation to furnish the proper basis for correlating brain and behavior mechanisms by specifying more adequately the structural and stimulus factors involved in such correlations.

BEHAVIOR WITH REFERENCE TO EXTRACEREBRAL MECHANISMS

Finally, I shall discuss some of the baffling problems that arise when dealing with forms and alterations of behavior which apparently can be understood only by assuming that conditions in the extracerebral environment exert an influence on the brain although the manner in which such an influence is exerted remains obscure. There is abundant evidence from anatomical, physiological, biochemical, and pathological investigations that various extracerebral factors influence the brain and thus become instrumental in bringing about behavior alterations (67). However, problems of particular interest arise when dealing with the exchange of substances between the blood and central nervous system. Experiments involving the use of drugs, viruses, dyes, and toxins have definitely established the existence of a blood-brain barrier which is impermeable or relatively impermeable to certain substances injected into the vascular system. The problem which concerns us is how substances circulating in the blood can produce profound behavior changes although they cannot be identified in the brain and apparently do not reach the central nervous system. Instead of trying to answer this question, I shall be content with calling attention to some of the relevant factors involved by citing the results of recent experiments with gold compounds.

After the injection of gold compounds, such as sanocrysin, into animals of different species, Querido (63) and others found that the cells of all tissues and organs show black specks and granules if the sections, previous to staining, are exposed to strong light and a "physical developer" containing silver nitrate. Microscopical examination does not reveal any difference between the black particles in nerve and glia cells and those in cells of other tissues. According to Querido, the formation of such particles in cells of the central nervous system is due to factors entirely different from those operative in cells outside the central nervous system (including the cells of the spinal ganglia). On the basis of extensive experiments with mice, he concluded that the black specks in cells outside the central nervous system are due to a deposition of gold sulfide capable of attracting silver from the developer under the influence of light whereas those in cells of the central nervous system represent a colloidal "silver-lipoid combination" that is formed, even in the absence of light, when the developer is applied. Although no gold or gold compound could be demonstrated in the central nervous system, injections of the gold compound produced not only neurological symptoms, such as ataxia, loss of orientation, and blindness, but also changes in the nerve cells (probably due to an increase in lipofuscin or a coalescence of finely dispersed droplets of lipides into larger drops). If there is an impermeability of the blood-brain barrier to gold, how, then, can gold poisoning affect the brain? Querido suggests that the metabolic disturbance in the brain is the result of a collateral or "parallel" poisoning; that is to say, the injected gold compound causes a dysfunction in some other organ, such as the liver or kidney, which in turn produces a change affecting the cells in the central nervous system.

Returning to the field of behavior, I submit that many behavior changes and perhaps some of the psychoses may well be the result of collateral poisonings. While the genesis of certain behavior alterations may lie in the extracerebral environment, the form and structure of such behavior cannot necessarily be understood without reference to cerebral mechanisms. To say that behavior may be viewed as a chemical epiphenomenon of events occurring in the extracerebral environment does not imply that the particular manifestations of behavior must be entirely independent of the brain and its mechanisms. (Similarly, the fact that the *hallucinations pédonculaires* described by French neurologists are brought about by lesions in the peduncles does not imply that the structure and form of the hallucinosis can be understood without recourse to the activity of the cerebral cortex.)

The difficulties involved in analyzing the relations between extracerebral factors and the brain have recently been brought home to me in trying to understand the behavior alterations associated with disturbances of porphyrin metabolism. Normally, the brain contains only small amounts of porphyrin (44–46). On discovering that the fluorescence spectrum of the white matter of the central nervous system reveals a sharp emission band with a maximum at about 625 millimicrons, I made various attempts to extract and identify the porphyrins occurring in the white matter. With the ethyl acetate-glacial acetic acid technique, I found that the myelinated fiber masses of the brain and spinal cord contain chiefly coproporphyrin and, on the basis of preliminary results, I concluded that it is the type III isomer which is present in the central nervous system. While the 625-millimicron emission band was found to be present in the fluorescence spectrum of the white matter in all warm-blooded animals (forty-three species of mammals and birds), no evidence was obtained for the occurrence of coproporphyrin or any other porphyrin in the peripheral nervous system. The fluorescence spectra of the cranial nerves exhibit characteristic differences in the sense that the 625-millimicron emission band is present in some nerves, such as the optic and auditory nerves, and absent in others, such as the oculomotor and abducens nerves. In the trigeminal nerve it is present only in the central portion of the nerve trunk. When the central portion showing this band is separated from the peripheral portion by cutting the nerve, histological study of these portions indicates that such a cut separates the glial from the non-glial portion of the nerve. Figure 10 represents a longitudinal section through the central portion of the trigeminal nerve of the rhesus monkey stained by using Dockrill's modification of del Río-Hortega's silver carbonate method for oligodendroglia (62). The central portion was obtained by cutting off the distal part that did not exhibit the porphyrin fluorescence spectrum. Figure 11 shows the central glial and peripheral non-glial segments and the glial dome as seen in the uncut trigeminal nerve of the rhesus monkey. A detailed study of the cranial nerves in various mammals, including man, led to the conclusion that the porphyrin fluorescence spectrum is characteristic only of the central glial segments and that the junction between the central and peripheral nervous system in the cranial nerve trunks separates, therefore, a region in which the 625-millimicron band is present from a region in which this band is absent. Various lines of evidence strongly suggest that the occurrence of coproporphyrin in the white matter of the central nervous system and in the central glial segments of cranial nerve trunks is correlated with the presence of oligodendroglia.

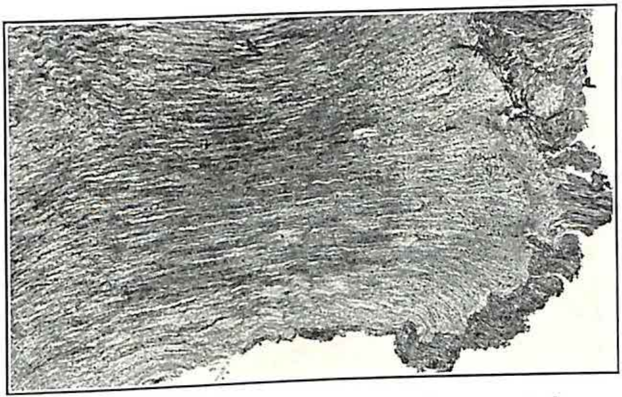


FIGURE 10. Photomicrograph of a section through the trigeminal nerve of a rhesus monkey. Impregnation with silver carbonate (Dockrill's modification of Río-Hortega's method.) ×41.

A different picture presents itself in disorders of porphyrin metabolism. It is well known that in the intermittent acute form of porphyria the patients frequently exhibit a large variety of nervous and mental symptoms. The neurological and psychiatric manifestations are of such a nature that the clinical picture is often confused with that of other neuropsychiatric disorders. Porphyria patients have frequently been diagnosed and treated as cases of acute encephalitis, encephalitis lethargica, poliomyelitis, myelitis, progressive muscular atrophy, polyneuritis, ascending acute polyneuritis, periarteritis nodosa, nicotine poisoning, arsenic poisoning, diphtheria, brain tumor, hysteria, vegetative neurosis, psychoneurosis, neurasthenia, acute psychosis, or manic-depressive psychosis (78, 79). Many of the symptoms observed in the intermittent acute type of porphyria point, therefore, to an involvement of the central nervous system.

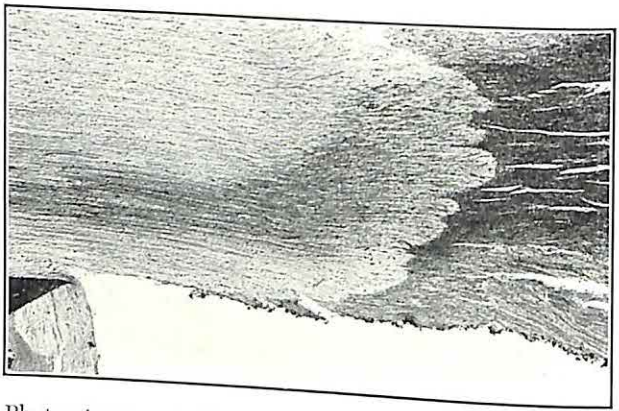


FIGURE 11. Photomicrograph of a section through the trigeminal nerve of a rhesus monkey. Impregnation with silver carbonate (Dockrill's modification of Río-Hortega's method.) ×25.

Biochemical studies, particularly by Watson and his co-workers in this country (24, 86), have been chiefly concerned with isolating the uroporphyrins occurring in the urine and feces of porphyria patients. In studying the occurrence and distribution of porphyrins in various organs and tissues obtained at necropsy from porphyria patients, I found that the blood serum and practically all non-nervous organs and tissues contain ether-soluble and/or ether-insoluble porphyrins. In fact, a spectroscopic examination of the "primary fluorescence," that is, the fluorescence which appears without previous treatment with chemical agents, reveals a characteristic porphyrin fluorescence spectrum in almost all non-nervous organs and tissues. In the adrenal cortex, for instance, the porphyrins are confined to the zona reticularis. Figure 12 shows the photomicrograph of an unstained frozen section through the adrenal cortex of a porphyria case. In such sections, only the zona reticularis (the dark zone in Figure 12) exhibits a red fluorescence and a porphyrin fluorescence spectrum. Porphyrins may also occur in the peripheral nervous system in certain stages of the disease. The ventral and dorsal roots as well as the spinal nerves may exhibit a pronounced red fluorescence and a fluorescence spectrum with a sharp and well-defined emission band at about 618 millimicrons. Different portions of a given spinal nerve may differ markedly with respect to the intensity of the 618-millimicron band. This band and the red fluorescence may be absent in some spinal nerves or in certain segments of a given nerve. These findings suggest that the porphyrins invade the peripheral nervous system in an irregular and patchy fashion.

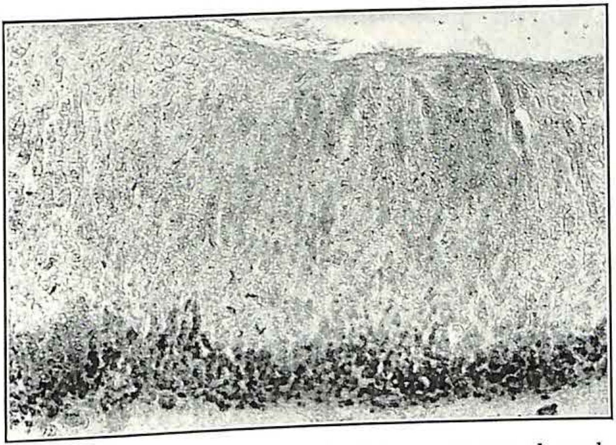


FIGURE 12. Photomicrograph of an unstained frozen section through the adrenal cortex of a porphyria case. ×31.

In view of the fact that many of the symptoms present in porphyria patients point to an involvement of the central nervous system, it is significant that the brains and spinal cords contain only the small amounts of normally occurring porphyrins. The data obtained so far indicate, therefore, that the blood-brain barrier is impermeable to the porphyrins circulating in the blood of cases of intermittent acute porphyria. Since the disease does not produce an abnormal "porphyrinization" of the brain, the symptoms referable to the central nervous system cannot be understood on the basis of a "cerebral porphyria." In passing, I should like to mention that so far I have not succeeded experimentally in definitely increasing or decreasing the concentration of the normally occurring porphyrins in the brain of animals. The procedures included injections of porphyrins and the production of nutritional deficiencies. Nor have I been able to find a disease affecting the porphyrin level in the central nervous system. For instance, extractions of the brains of cases of dementia praecox, senile dementia, Jacksonian epilepsy, or cerebral arteriosclerosis furnished only the small amounts of normally occurring porphyrins. In fact, there is no clear-cut evidence that even any of the diseases involving disorders of porphyrin metabolism or any of the chemically induced porphyrias or coproporphyrinurias are associated with a "porphyrinization" of the brain. Furthermore, the occurrence of porphyrins in nerve and glia cells has never been microscopically demonstrated in brains of cases of intermittent acute porphyria or in brains of cases of the congenital (photosensitive) type of porphyria.

The results obtained in studying cases of intermittent acute porphyria indicate, therefore, that the porphyrins characteristic of this disease do not reach the central nervous system. At the same time, the symptoms referable to the central nervous system are undoubtedly associated with a disorder of porphyrin metabolism. It will be a matter of future research to specify the mechanisms involved in producing these symptoms. In the meantime, certain facts and considerations seem pertinent in attempting to account for the behavior alterations which point to an involvement of the central nervous system.

Some investigators have reported histopathological alterations in the brain while others have been unable to find such changes. It is difficult to understand how such histopathological changes as have been reported can account, for instance, for the complex symptomatology of a psychosis as observed in acute porphyria. In the absence of histopathological alterations it may be argued, of course, that the cellular changes are reversible or not detectable with the methods available at the present time. However, falling back on reversible or undetectable changes merely shifts the problem and is of no help in specifying the factors responsible for the alteration in brain functions.

In specifying such factors, account must be taken of the fact that the application of chromatographic techniques provides evidence that certain body fluids of porphyria patients contain, in addition to previously described porphyrins, a number of unidentified porphyrins as well as other pigments. Intermittent acute porphyria, therefore, may involve general metabolic disturbances as well as a disorder of porphyrin metabolism. The pigments, however, have not been found in the brains of the porphyria patients I have studied. It is possible, of course, that the production of various pigments leads to or is accompanied by the production of substances capable of affecting or invading the brain. In such a case, the psychiatric manifestations of the disease could be considered the result of a "collateral poisoning." In the meantime it is not even known whether a substance such as porphobilinogen is present in the brain of porphyria cases. Porphyrins, however, invade the endocrines. Since published reports indicate that porphyrin injections lead to an increased secretion of gonadotropic and melanophore-dispersing hormones (27, 28, 31, 66) and certain changes in intermediary metabolism (29, 30), alterations in the hormone production of various endocrines may be a characteristic feature of intermittent acute porphyria. In fact, it has been suggested that the clinical manifestations of this disease are chiefly brought about by a vasoconstrictor substance circulating in the blood (15).

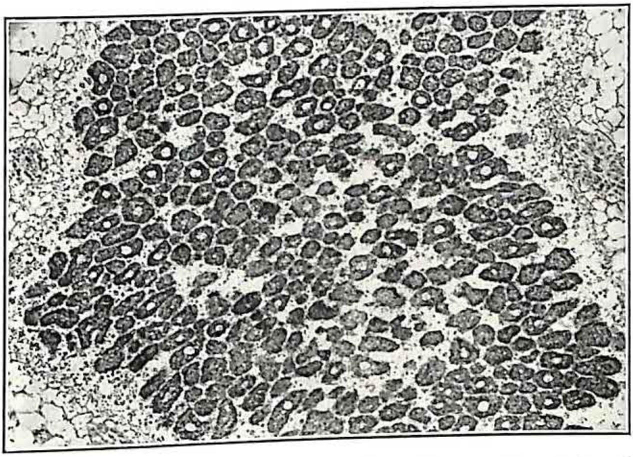


FIGURE 13. Photomicrograph of a section through a root nodule of *Phaseolus vulgaris*. Modification of Masson's method for argentophil cells.

It must be emphasized at this point that the functions of the free porphyrins in the organism have been little studied. The physiological role even of the naturally occurring coproporphyrins is not exactly known. Furthermore, the relations between free porphyrins and hemoglobin are complicated by numerous factors. For this reason I searched for a porphyrin-hemoglobin system that might be studied in the absence of factors complicating or influencing the interrelations of porphyrins and hemoglobin metabolism in animals. Starting from Kubo's discovery (53) that the root nodules of leguminous plants contain hemoglobin, I found that hemoglobin containing nodules when crushed exhibit a reddish fluorescence and a fluorescence spectrum with a well-defined emission band at about 620 millimicrons. Subsequently I was able to isolate various porphyrins from root nodule material (47, 48). Figure 13 represents a photomicrograph of a section through a root nodule of *Phaseolus vulgaris* (var. Red Kidney bean). In unstained frozen sections, red fluorescence and a porphyrin fluorescence spectrum can be seen only in the cytoplasm of the large cells containing *Rhizobium* (Figure 14). There is further evidence that the leghemoglobin is present in the cytoplasm of the same cells. Porphyrins, hemoglobin, and bacteria, therefore, have the same distribution in the nodule cells. The simultaneous occurrence of hemoglobin and porphyrins in the root nodules of leguminous plants is of fundamental interest in view of the fact that these substances occur here in a system in which living cells are capable of fixing atmospheric nitrogen. It is hoped that further research on the porphyrins in the root nodules will throw new light on the physiological significance of porphyrins in general.

In closing, I should like to refer to a statement made by A. J. Clark in discussing the principles and methods of a quantitative pharmacology (11). Speaking of the complexity of living cells he expressed the view "that, if a reaction between a drug and a cell appears to follow simpler laws than those applicable to relatively simple colloidal systems, this apparent simplicity is an accident. . . ." I feel tempted to voice the opinion that for a long time to come—and by "a long time" I do not mean, like Dr. von Neumann, four or five years, but forty or fifty years or even centuries—the laws of psychology will not even be as simple as those applicable to relatively simple colloidal systems. If "laws" of such simplicity will be found in the near future they will not be *psychological* laws, but carefully contrived "accidents" attained by ignoring relevant facts.

REFERENCES

1. Allen, W. F. Effect of ablating the frontal lobes, hippocampi, and occipito-parieto-temporal (excepting pyriform areas) lobes on positive and negative olfactory conditioned reflexes. *Amer. J. Physiol.*, 1940, **128**, 754–771.
2. Allen, W. F. Effect of ablating the pyriform-amygdaloid areas and hippocampi on positive and negative olfactory conditioned reflexes and on conditioned olfactory differentiation. *Amer. J. Physiol.*, 1941, **132**, 81–92.
3. Allen, W. F. Degeneration in the dog's mammillary body and Ammon's horn following transection of the fornix. *J. Comp. Neurol.*, 1944, **80**, 283–291.
4. Allen, W. F. Fiber degeneration in Ammon's horn resulting from extirpations of the piriform and other cortical areas and from transection of the horn at various levels. *J. Comp. Neurol.*, 1948, **88**, 425–438.
5. Bard, P., and Mountcastle, V. B. Some forebrain mechanisms involved

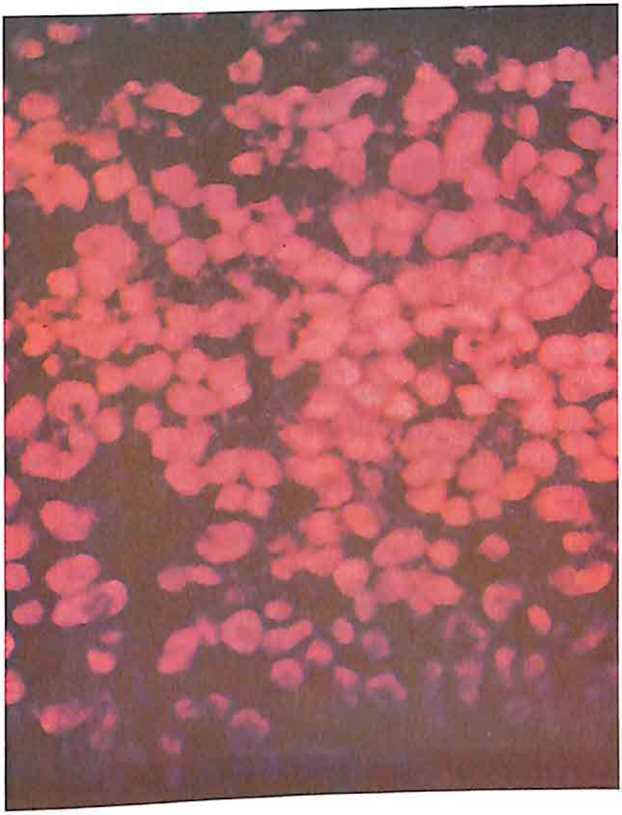


FIGURE 14. Photomicrograph of an unstained frozen section cut at 25 microns through a root nodule of *Phaseolus vulgaris* (var. Red Kidney bean), showing the red fluorescence of the cells under near-ultraviolet light.

in expression of rage with special reference to suppression of angry behavior. *Res. Publl. Assoc. Res. Nerv. & Ment. Dis.*, 1948, **27**, 362–404.

6. Brodal, A. The hippocampus and the sense of smell. *Brain*, 1947, **70**, 179–222.
7. Brookhart, J. M., Dey, F. L., and Ranson, S. W. The abolition of mating behavior by hypothalamic lesions in guinea pigs. *Endocrinology*, 1941, **28**, 561–565.
8. Brown, S., and Schäfer, E. A. An investigation into the functions of the occipital and temporal lobes of the monkey's brain. *Phil. Trans. Roy. Soc. London*, 1888, **179B**, 303–327.
9. Bucy, P. C., and Klüver, H. Anatomic changes secondary to temporal lobectomy. *Arch. Neurol. & Psychiat.*, 1940, **44**, 1142–1146.
10. Chaikoff, I. L., Reichert, F. L., Larson, P. S., and Mathes, M. E. The effect of hypophysectomy and cerebral manipulation in the dog upon the response of the blood sugar and inorganic phosphorus to insulin. *Amer. J. Physiol.*, 1935, **112**, 493–503.
11. Clark, A. J. *General pharmacology.* Vol. IV of Ergänzungswerk of Heffter's *Hdb. d. exp. Pharmakol.*, ed. by W. Heubner and J. Schüller. Berlin: Springer, 1937.
12. Degkwitz, R. *Lipoide und Ionen.* Dresden: Steinkopff, 1933.
13. De Jong, H. Die experimentelle Katatonie als vielfach vorkommende Reaktionsform des Zentralnervensystems. *Zsch. f. d. ges. Neurol. u. Psychiat.*, 1932, **139**, 468–499.
14. De Jong, H. L. *Experimental catatonia.* Baltimore: Williams & Wilkins Co., 1945.
15. Denny-Brown, D., and Sciarra, D. Changes in the nervous system in acute porphyria. *Brain*, 1945, **68**, 1–16.
16. Dey, F. L. Changes in ovaries and uteri in guinea pigs with hypothalamic lesions. *Amer. J. Anat.*, 1941, **69**, 61–87.
17. Dey, F. L. Evidence of hypothalamic control of hypophyseal gonadotropic functions in the female guinea pig. *Endocrinology*, 1943, **33**, 75–82.
18. Dey, F. L., Fisher, C., Berry, C. M., and Ranson, S. W. Disturbances in reproductive functions caused by hypothalamic lesions in female guinea pigs. *Amer. J. Physiol.*, 1940, **129**, 39–46.
19. Dey, F. L., Fisher, C., and Ranson, S. W. Disturbances in pregnancy and labor in guinea pigs with hypothalamic lesions. *Amer. J. Obstet. & Gynecol.*, 1941, **42**, 459–466.
20. Dey, F. L., Leininger, C. R., and Ranson, S. W. The effect of hypophysial lesions on mating behavior in female guinea pigs. *Endocrinology*, 1942, **30**, 323–326.
21. Ferraro, A., and Barrera, S. E. *Experimental catalepsy.* Utica: State Hospitals Press, 1932.
22. Ferrier, D. Schäfer on the temporal and occipital lobes. *Brain*, 1888, **11**, 7–30.
23. Fränkel, S. *Die Arzneimittel-Synthese auf Grundlage der Beziehungen zwischen chemischem Aufbau und Wirkung.* Berlin: Springer, 1927.
24. Grinstein, M., Schwartz, S., and Watson, C. J. Studies of the uroporphyrins. I. The purification of uroporphyrin I and the nature of Waldenström's uroporphyrin, as isolated from porphyria material. *J. Biol. Chem.*, 1945, **157**, 323–343.
25. Gunn, J. A. Relations between chemical constitution, pharmacological actions, and therapeutic uses, in the harmine group of alkaloids. *Arch. internat. de pharmacodyn. et de thérap.*, 1935, **50**, 379–396.

26. Hartung, W. H. Epinephrine and related compounds: influence of structure on physiological activity. *Chem. Rev.*, 1931, **9**, 389–465.
27. Hinsberg, K. Über den Einfluss der Porphyrine auf die Hypophyse. *Klin. Wchnschr.*, 1939, **18**, 180.
28. Hinsberg, K. Endogene und exogene Ursachen bei der Krebsentstehung. *Dtsch. med. Wchnschr.*, 1940, **66**, 1074–1078.
29. Hinsberg, K., and Merten, R. Über die Wirkung der Porphyrine auf den Stoffwechsel des Warmblüters. *Bioch. Zsch.*, 1938, **299**, 137–140.
30. Hinsberg, K., and Merten, R. Über die Wirkung des Porphyrins auf den Stoffwechsel des Warmblüters. II. *Bioch. Zsch.*, 1939, **302**, 103–111.
31. Hinsberg, K., and Rodewald, W. Über die Wirkung von Porphyrin auf die Hypophyse, besonders auf die Melanophorenhormonausschüttung. *Arch. f. exp. Pathol. u. Pharmakol.*, 1939, **191**, 1–11.
32. Kleist, K. Bericht über die Gehirnpathologie in ihrer Bedeutung für Neurologie und Psychiatrie. *Zsch. f. d. ges. Neurol. u. Psychiat.*, 1937, **158**, 159–193.
33. Klüver, H. *Mescal*. London: Kegan Paul, Trench, Trubner & Co., 1928.
34. Klüver, H. The equivalence of stimuli in the behavior of monkeys. *J. Genet. Psychol.*, 1931, **39**, 3–27.
35. Klüver, H. *Behavior mechanisms in monkeys*. Chicago: University of Chicago Press, 1933.
36. Klüver, H. A tachistoscopic device for work with sub-human primates. *J. Psychol.*, 1935, **1**, 1–4.
37. Klüver, H. Use of vacuum tube amplification in establishing differential motor reactions. *J. Psychol.*, 1935, **1**, 45–47.
38. Klüver, H. The study of personality and the method of equivalent and non-equivalent stimuli. *Character and Personality*, 1936, **5**, 91–112.
39. Klüver, H. An analysis of the effects of the removal of the occipital lobes in monkeys. *J. Psychol.*, 1936, **2**, 49–61.
40. Klüver, H. Certain effects of lesions of the occipital lobes in macaques. *J. Psychol.*, 1937, **4**, 383–401.
41. Klüver, H. Visual functions after removal of the occipital lobes. *J. Psychol.*, 1941, **11**, 23–45.
42. Klüver, H. Mechanisms of hallucinations. Chapter X (pp. 175–207) in *Studies in personality*. New York: McGraw-Hill Book Co., 1942.
43. Klüver, H. Functional significance of the geniculo-striate system. *Biol. Symposia*, 1942, **7**, 253–299.
44. Klüver, H. Porphyrins, the nervous system, and behavior. *J. Psychol.*, 1944, **17**, 209–228.
45. Klüver, H. Porphyrins and the central nervous system. *Federation Proceedings*, 1944, **3**, 25–26.
46. Klüver, H. On naturally occurring porphyrins in the central nervous system. *Science*, 1944, **99**, 482–484.
47. Klüver, H. On a possible use of the root nodules of leguminous plants for research in neurology and psychiatry (preliminary report on a free porphyrin-hemoglobin system). *J. Psychol.*, 1948, **25**, 331–356.
48. Klüver, H. On naturally occurring porphyrins in the root nodules of leguminous plants. *Federation Proceedings*, 1948, **7**, 66.
49. Klüver, H., and Bucy, P. C. "Psychic blindness" and other symptoms following bilateral temporal lobectomy in rhesus monkeys. *Amer. J. Physiol.*, 1937, **119**, 352–353.

50. Klüver, H., and Bucy, P. C. An analysis of certain effects of bilateral temporal lobectomy in the rhesus monkey, with special reference to "psychic blindness." *J. Psychol.*, 1938, **5**, 33–54.
51. Klüver, H., and Bucy, P. C. A preliminary analysis of the functions of the temporal lobes in monkeys. *Trans. Amer. Neurol. Assoc.*, 1939, pp. 170–175.
52. Klüver, H., and Bucy, P. C. Preliminary analysis of functions of the temporal lobes in monkeys. *Arch. Neurol. & Psychiat.*, 1939, **42**, 979–1000.
53. Kubo, H. Über das Hämoprotein aus den Wurzelknöllchen von Leguminosen. *Acta Phytochimica*, 1939, **11**, 195–200.
54. Leininger, C. R., and Ranson, S. W. The effect of hypophysial stalk transection upon gonadotrophic function in the guinea pig. *Anat. Rec.*, 1943, **87**, 77–83.
55. Meyer, A. The Wernicke syndrome. *J. Neurol., Neurosurg. & Psychiat.*, 1944, **7** (new series), 66–75.
56. Monakow, C. v., and Mourgue, R. *Biologische Einführung in das Studium der Neurologie und Psychopathologie.* Stuttgart and Leipzig: Hippokrates-Verlag, 1930.
57. Murphy, J. P., and Gellhorn, E. Further investigations on diencephalic-cortical relations and their significance for the problem of emotion. *J. Neurophysiol.*, 1945, **8**, 431–448.
58. Noteboom, L. Experimental catatonia by means of derivatives of mescaline and adrenaline. *Proc. Kon. Akad. v. Wetensch. Amsterdam*, 1934, **37**, No. 8.
59. Obrador, S. Temporal lobotomy. *J. Neuropathol. & Exp. Neurol.*, 1947, **6**, 185–193.
60. Obrador, S. *Las modernas intervenciones quirúrgicas en psiquiatría.* Madrid: Editorial Paz Montalvo, 1947.
61. Papez, J. W. A proposed mechanism of emotion. *Arch. Neurol. & Psychiat.*, 1937, **38**, 725–743.
62. Penfield, W. A further modification of del Río-Hortega's method of staining oligodendroglia. *Amer. J. Pathol.*, 1930, **6**, 445–448.
63. Querido, A. Gold intoxication of nervous elements. On the permeability of the blood-brain barrier. *Acta psychiat. neurol.*, 1947, **22**, 97–151.
64. Renshaw, B., Forbes, A., and Morison, B. R. Activity of isocortex and hippocampus: electrical studies with micro-electrodes. *J. Neurophysiol.*, 1940, **3**, 74–105.
65. Richter, C. P. Total self regulatory functions in animals and human beings. *Harvey Lect.*, 1942–1943, **38**, 63–103.
66. Rodewald, W. Über die oestrogene Wirkung von Porphyrinen. *Arch. f. exp. Pathol. u. Pharmakol.*, 1940, **194**, 76–77.
67. Roizin, L. Histometabolic changes and neuropathologic selectivity in the light of recent investigations. *J. Neuropathol. & Exp. Neurol.*, 1948, **7**, 216–233.
68. Rose, J. E., Woolsey, C. N., and Jarcho, L. W. Relations of anterior thalamic nuclei and mammillothalamic tract to limbic cortex. *Federation Proceedings*, 1947, **6**, 193.
69. Schäfer, E. A. Experiments on special sense localisations in the cortex cerebri of the monkey. *Brain*, 1888, **10**, 362–380.
70. Schäfer, E. A. On the functions of the temporal and occipital lobes: a reply to Dr. Ferrier. *Brain*, 1888, **11**, 145–165.
71. Smith, W. K. The results of ablation of the cingular region of the cerebral cortex. *Federation Proceedings*, 1944, **3**, 42–43.

72. Smith, W. K. The results of stimulation of the uncus and adjacent portions of the hippocampal gyrus. *Federation Proceedings*, 1944, **3**, 43.
73. Smith, W. K. The functional significance of the rostral cingular cortex as revealed by its responses to electrical excitation. *J. Neurophysiol.*, 1945, **8**, 241–255.
74. Speransky, A. D. *A basis for the theory of medicine.* New York: International Publishers, 1935.
75. Spielmeyer, W. Von den Beziehungen zwischen Substrat und Funktion. *Zsch. f. d. ges. Neurol. u. Psychiat.*, 1930, **127**, 777–782.
76. Spitzer, A. Anatomie und Physiologie der zentralen Bahnen des Vestibularis. *Arbeiten a. d. Neurol. Inst. a. d. Wiener Universität*, 1924, **25**, 423–470.
77. Stoll, W. A. Lysergsäure-diäthylamid, ein Phantastikum aus der Mutterkorngruppe. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 1947, **60**, 279–323.
78. Waldenström, J. Studien über Porphyrie. *Acta Med. Scand.*, 1937, suppl. **82**, 1–254.
79. Waldenström, J. Neurological symptoms caused by so-called acute porphyria. *Acta psychiat. neurol.*, 1939, **14**, 375–379.
80. Walker, A. E. An experimental study of the thalamo-cortical projection of the macaque monkey. *J. Comp. Neurol.*, 1936, **64**, 1–39.
81. Walker, A. E. The projection of the medial geniculate body to the cerebral cortex in the macaque monkey. *J. Anat.*, 1937, **71**, 319–331.
82. Walker, A. E. *The primate thalamus.* Chicago: University of Chicago Press, 1938.
83. Ward, A. A., Jr. The anterior cingulate gyrus and personality. *Res. Publ. Assoc. Res. Nerv. & Ment. Dis.*, 1948, **27**, 438–445.
84. Ward, A. A., Jr. The cingular gyrus: area 24. *J. Neurophysiol.*, 1948, **11**, 13–23.
85. Ward, A. A., Jr., and McCulloch, W. S. The projection of the frontal lobe on the hypothalamus. *J. Neurophysiol.*, 1947, **10**, 309–314.
86. Watson, C. J., Schwartz, S., and Hawkinson, V. Studies of the uroporphyrins. II. Further studies of the porphyrins of the urine, feces, bile, and liver in cases of porphyria, with particular reference to a Waldenström type porphyrin behaving as an entity in the Tswett column. *J. Biol. Chem.*, 1945, **157**, 345–361.
87. Wertham, F., and Wertham, F. *The brain as an organ.* New York: The Macmillan Co., 1934.
88. Woolsey, C. N., Dick, F. M., and Frantz, R. H. Electrical responses in gyrus cinguli evoked by electrical stimulation of ipsilateral mammillary body in cat and monkey. *Federation Proceedings*, 1946, **5**, 116.

DISCUSSION

DR. BROSIN: Dr. Nielsen has some provocative cases of interest in this connection, together with slides, and I think we might very well have them now before the formal discussion.

DR. NIELSEN: I noticed in Dr. Klüver's film that his monkeys didn't have complete visual agnosia. They recognized the stepladder; they recognized the hole they jumped into; they seemed to know what was safe to put their feet on, and jumped the distance up to the hole. Is that correct?

DR. KLÜVER: I think I was careful not to say that the monkeys had a visual agnosia; I merely said that they exhibited forms of behavior which appear to indicate a visual agnosia or psychic blindness. The animals had apparently no difficulty in discriminating visually. If I were to employ the terminology previously used by clinical writers and which you have so expertly analyzed in your book, *Agnosia, Apraxia, Aphasia,* I would say that the monkeys exhibited the picture of an "associative mind-blindness," as described by Lissauer in 1890. The type of disturbance present in these monkeys is certainly not Lissauer's "apperceptive mind-blindness."

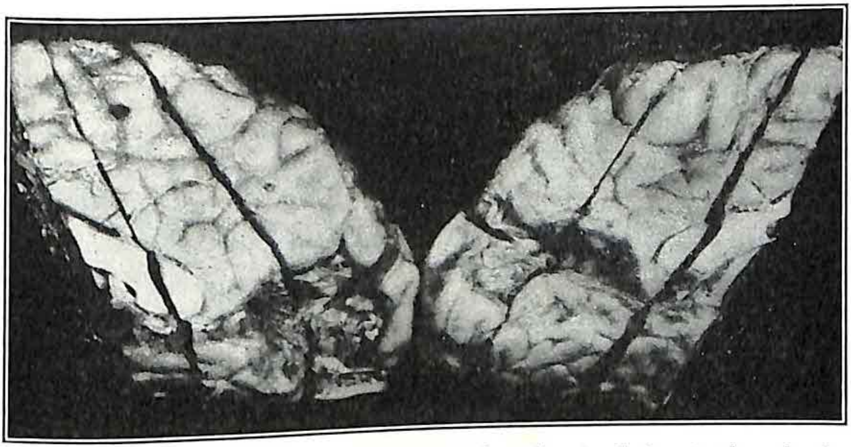


FIGURE 1. Mesial surfaces of both occipital lobes, showing lesions in the calcarine areas of both sides. (After J. M. Nielsen. *Agnosia, Apraxia, Aphasia.* New York: Paul B. Hoeber, 1946, 2nd Edition, p. 179. By permission of the publishers.)

DR. NIELSEN: This is a most marvelous demonstration of Dr. Klüver's work. I just wanted to make sure that we were not discussing different things, because the few slides that I have selected to show in the short time available are of visual agnosia due to occipital lesions, and there is obviously a marked distinction between the function of the temporal lobe and the occipital lobe in man as compared to many of the lower animals. I don't want to have it interpreted that what I am showing here is to disprove anything previously presented in other talks here. It is simply that I want to present the facts, and then we can make comparisons.

These (Figure 1) are the mesial surfaces of both occipital lobes of a man who had emboli into the calcarine area of both sides. He immediately saw "a million stars," as he described it; and from that time on he was totally blind, but was able at all times to revisualize anything that he had formerly seen, and he was able to give visual descriptions, including colors, of all of his past memories. From this we concluded that, so far as visual memory is concerned, it does not reside in area 17; that is, not in the calcarine area, but in the association areas.

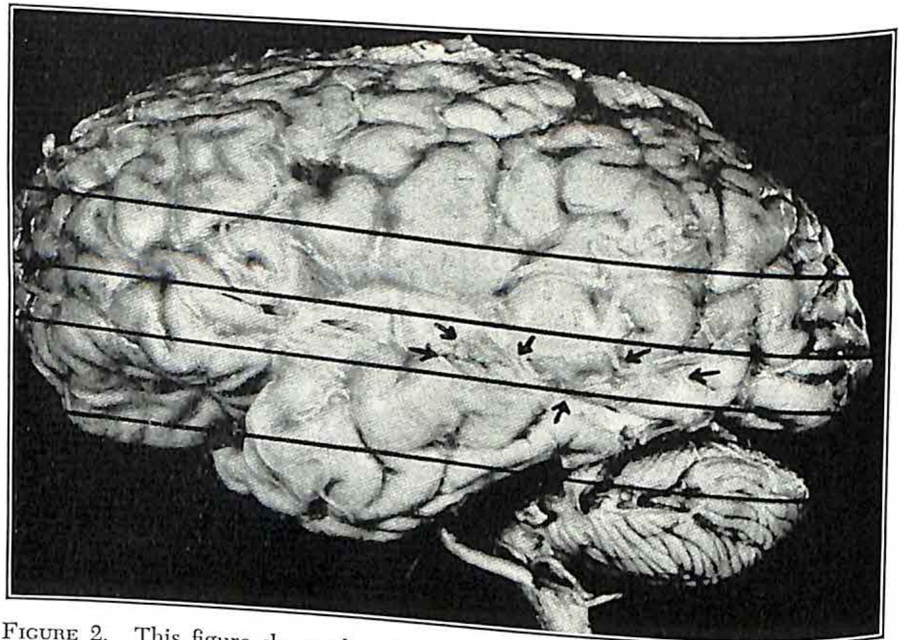


FIGURE 2. This figure shows the lateral view with lines of section. The sections are shown in Figure 3. Arrows indicate a band of softening. (After J. M. Nielsen. *Agnosia, Apraxia, Aphasia.* New York: Paul B. Hoeber, 1946, 2nd Edition, p. 180. By permission of the publishers.)

Here (Figure 2) is the lateral view of the brain of a man whom I had the opportunity to examine about two or three hundred times, since I saw him for several years. He had, in the course of his illness, a lesion of the left middle cerebral artery, particularly the angular gyrus, as is shown here. He was never again able to read, but did recover his capacity to speak, so there were no language disturbances in the later contacts with him. One morning I went to see him, and he said that he had had some trouble with his vision that morning. When I checked him, he had a *left* homonymous hemianopia, not a right. The lesion would have to be in the right occipital lobe or temporal lobe. He asked in detail about his relatives; he knew me; he knew his attorney; he knew all the people that he had known, including the nurses; but he did not know anything inanimate that he saw. He didn't *recognize* anything that he saw that was inanimate, nor could he *revisualize* anything that was inanimate. This presented a most striking picture. Of course, I didn't realize at first that this was going to be a categorical defect and that he would fail to recognize all inanimate objects. But as we went on he looked out of the window and asked me what "those things" were, indicating the outine of the mountains. (This was here in Pasadena.) I told him that they were the mountains. He replied, "Oh, they are mountains?" He asked, "What are those things down here?," indicating a row of automobiles. I said that they were automobiles. He said, "Oh, automobiles. Look at that mule grazing there." There was a mule in between the cars, and he recognized the mule. There was a very sharp line drawn between animate and inanimate objects. We had the opportunity to test flowers in his case also. I had a flower brought in without a word being said. When he saw it he said, "That's a daffodil, isn't it?" He included flowers with his animate objects. I would like to show you the horizontal sections of his brain, as made on the horizontal lines shown on this lateral view.

This (Figure 3*A*) shows a lesion placed in the right occipital lobe. We will ignore the signs of trouble in the left temporal lobe with which we are not concerned now, because it is not symbols we are dealing with; we are omitting aphasia from this discussion. The lesion in the right occipital lobe was in area 18.

This (Figure 3*B*) is one centimeter lower and shows a much more extensive lesion, completely undermining area 19 (the area for revisualization) although the cortex is intact.

Here (Figure 3*C*) we have further lesions in the same area, all on the right side. There is one small lesion on the left side, but it is not enough to give us any defect in the field of vision.

This (Figure 3*D*) was necessarily taken looking up, because the brain below this could not hang together, and this view is just for completeness of presentation. There is a lesion in the uncus of the hippocampal gyrus on the right side, which, so far as I know, was not related to this symptomatology at all, but it may have been related to some outbursts of anger the patient showed.

This (Figure 4) is an entirely different case. This was a woman who was operated on for lung abscess, and, as the operation was in progress, the abscess burst into the bronchus and almost suffocated her. Her life was just barely saved. She was then stuporous for a

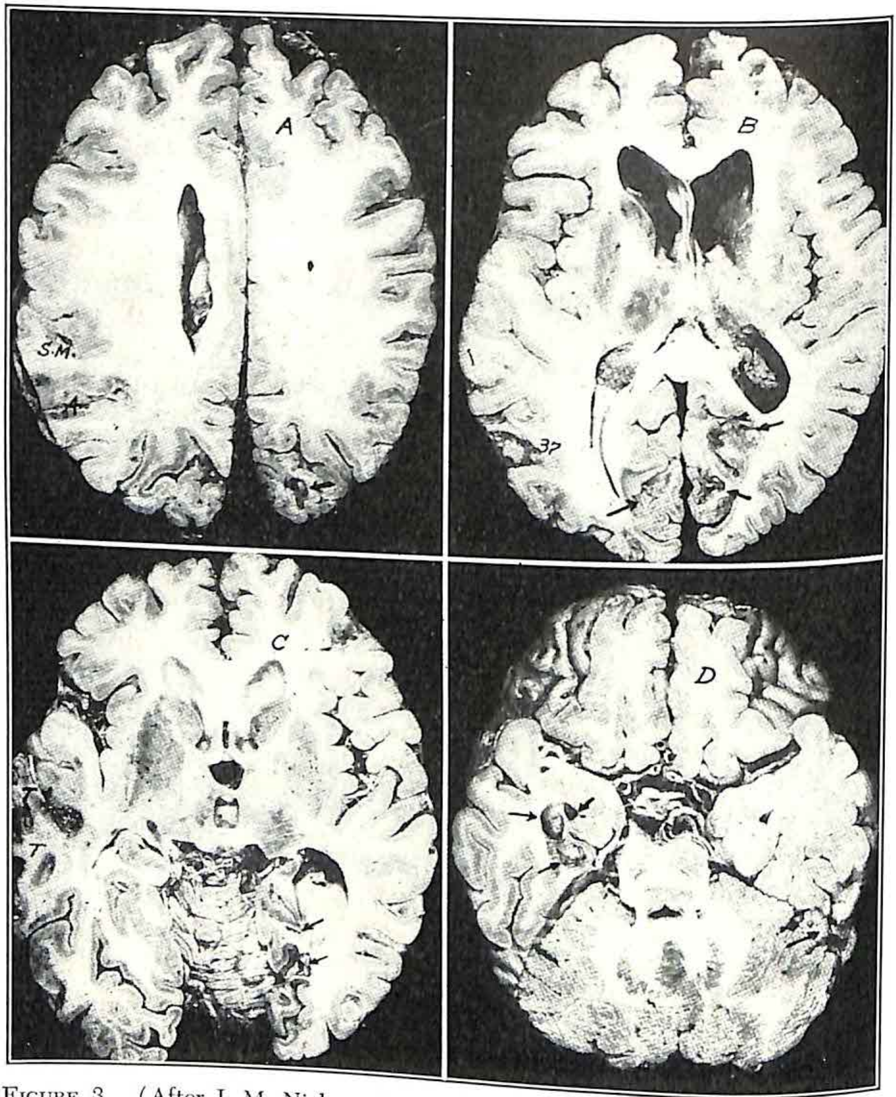
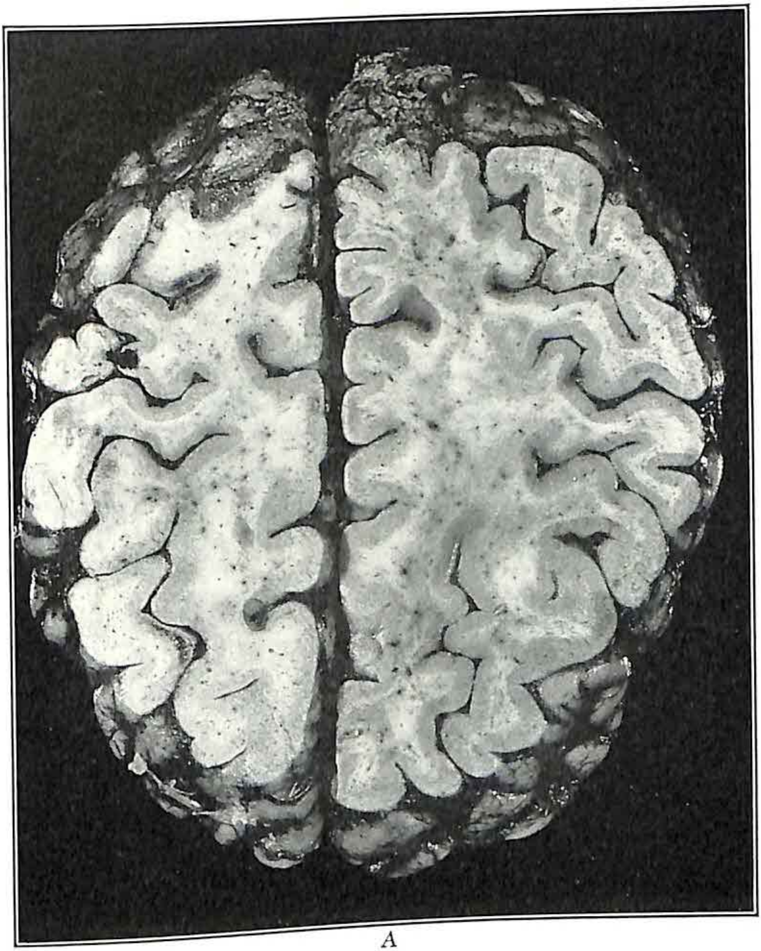


FIGURE 3. (After J. M. Nielsen. *Agnosia, Apraxia, Aphasia*. New York: Paul B. Hoeber, 1946, 2nd Edition, p. 181. By permission of the publishers.)

In *A* is seen subcortical softening in the left angular and supramarginal gyri. In the right occipital lobe is seen a lesion indicated by arrow (areas 18 and 19) placed so as to destroy completely the function of that structure. In *B* are seen three lesions, one cortical in the left area 37, the posterior extremity of the softening in the left temporal lobe. The second is a very small lesion in the left occipital lobe (arrow) cortical in area 17 (in the lingual gyrus), and hence affects the visual projection fiber system, not association tracts. The third (arrow) almost completely destroys the lingual gyrus on the right side, anteriorly destroying areas 18 and 19 also (association tracts). In *C* are seen three lesions. On the left side the first destroys the temporal lobe (*TT*) to within 1 cm. of the lateral ventricle (the anterior end escaping). The second destroys the lingual gyrus on the right anteriorly to the posterior horn of the lateral ventricle, which is dilated in consequence. This affects areas 18 and 19. The third is a small and insignificant lesion in the right prefrontal cortex. In *D* the photograph is taken looking up, and the cystic lesion (arrows) in the right hippocampus is on the left side of the figure. The slight cortical lesion of the left cerebellar lobe (arrow) is visible on the right side of the figure.

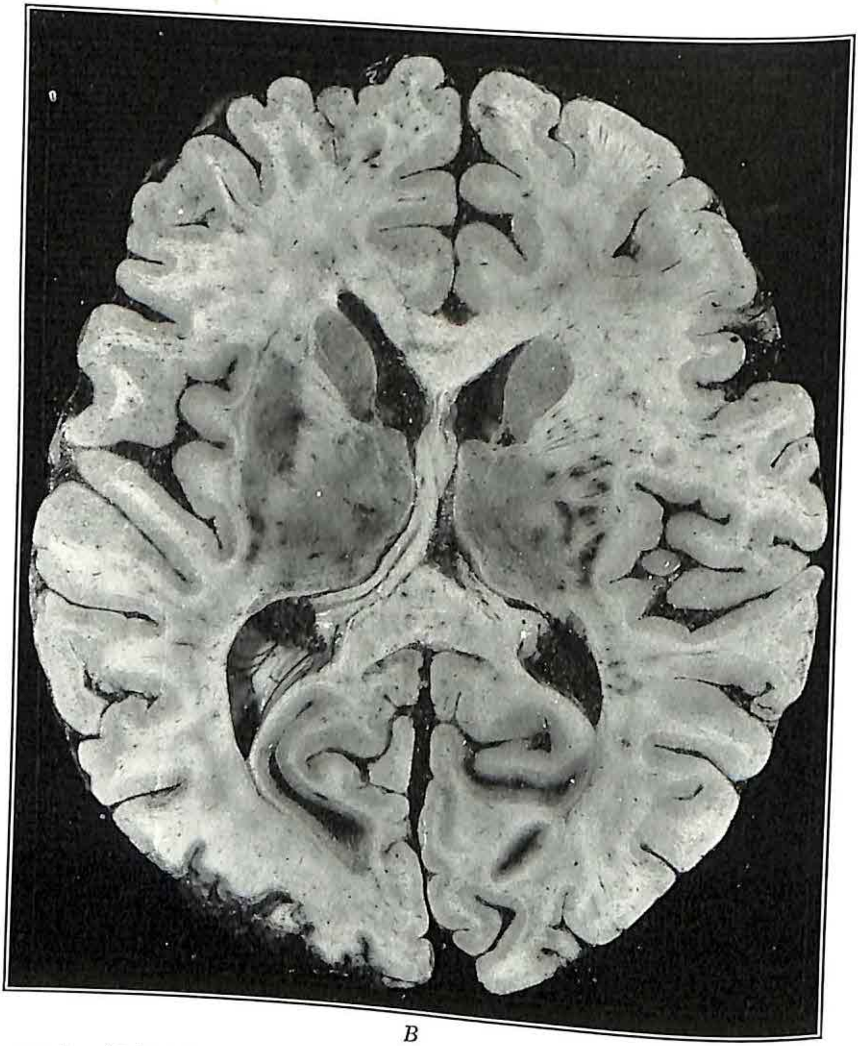
number of days. As she cleared, we began to examine her, tested her fields of vision by the confrontation test, and I asked her to tell me when she saw my finger moving. She never said anything. I again stimulated her and said, "Give me your attention. Look at my nose and tell me when my fingers move." Again she didn't say anything. I thought that perhaps my object was not bright enough, so I took a percussion hammer with a bright handle and held that out and she said, "I see it now." I then said, "Tell me when you see my finger." She never did see it. I held the hand immediately in front of her and said, "Don't you see my hand?" She said, "No." If I held an object



A

FIGURE 4. (*A*) This section shows a focal area of softening in each parietal lobe.

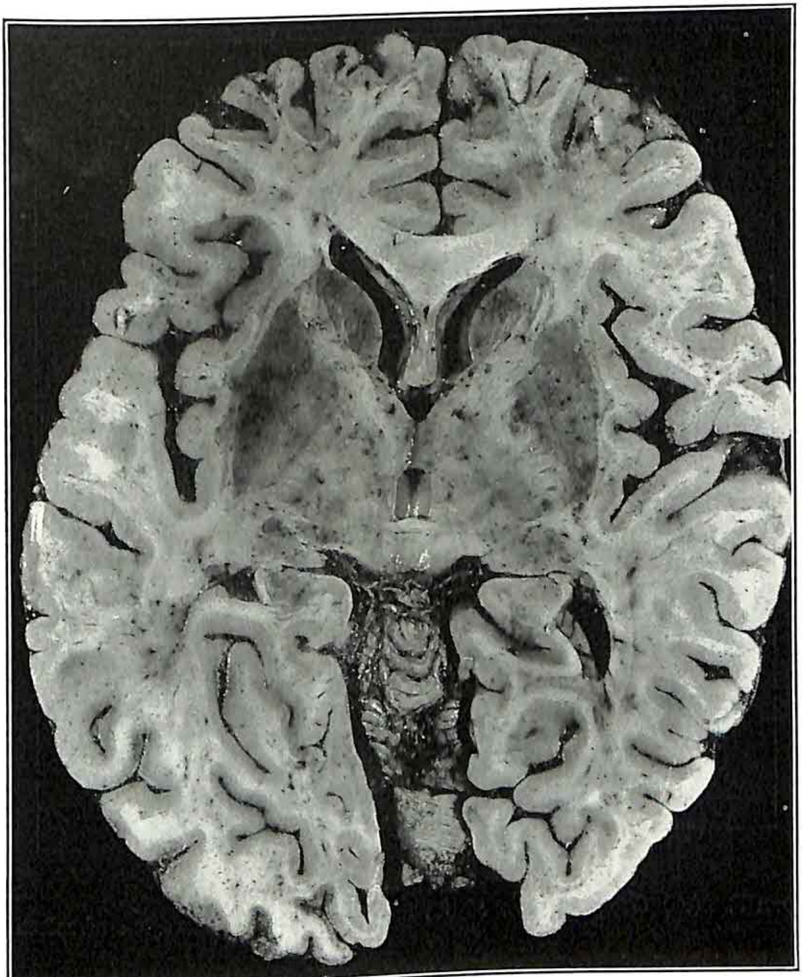
in front of her, she saw the object. It was some little time before I realized we had a complete case of visual agnosia for animate objects, not for inanimate objects. We held out a tray of objects including the



B

FIGURE 4. (*B*) This section shows cortical destruction of the left occipital lobe.

patient's artificial teeth. She picked up every object I asked for and handed it to me. When I asked for the teeth, she said there were no teeth. We cleared the table of everything but the teeth, and I asked her for them then, but she insisted that there were no teeth. She thought of the teeth as a part of animate objects. I asked the nurse to get a doll from the pediatrics department and held it up before her. It was a beautiful doll with artificial skin so that it felt very natural, and I held that up before her and asked, "What is this?" And she

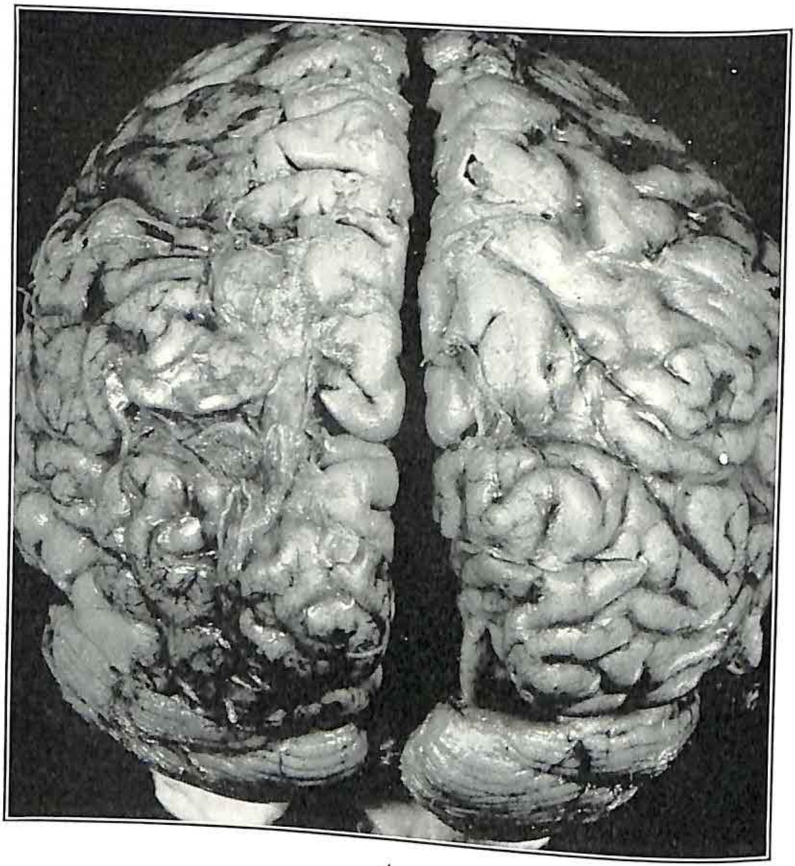


C

FIGURE 4. (*C*) This section also shows destruction of the left occipital lobe. (*A*, *B*, and *C* after J. M. Nielsen. *Agnosia, Apraxia, Aphasia*. New York: Paul B. Hoeber, 1946, 2nd Edition, p. 185. By permission of the publishers.)

said, "It's pink." I said, "Yes, it's pink, but what is the object?" She said, "pink," and that's as far as we could get. The three sections (Figure 4) show a decortication of the left occipital lobe.

The next case is illustrated by Figure 5*A*, *B*, *C*, *D*, and *E*. It is another case of complete visual agnosia for animate objects, but retention of recognition of everything inanimate. Again the lesion is in the left occipital lobe. Figure 5*A* is taken looking directly at the occipital lobe before the section was cut. You will see the lesion here, but as we proceed you will see how deeply it cuts into the lobe.



A

FIGURE 5. (*A*) Occipital view of the uncut brain, showing region of softening.

In Figure 5*B* we have the occipital lobe below, and it shows only the left half of the brain because the right was intact. There were no lesions in it. Here is the lesion cutting in rather deeply. In Figure 5*C* one can see the slit made by a vascular lesion which had occurred some weeks before.

Figure 5*D* shows some of the lesion remaining, but it isn't nearly as deep in this location. In Figure 5*E*, as we go down with the sections cut parallel, we can see there is still some disturbance but not very much; while in Figure 5*F* there is extremely little if any involvement at that level. Now, if we take cases like these and find that a right occipital lesion gives us a visual agnosia for inanimate objects, and the left gives visual agnosia for animate objects, you may think now that we have the system; but such localization is only an individual matter. There is no way of telling in advance in which occipital lobe the patterns are located. For that reason I have another case to present here.

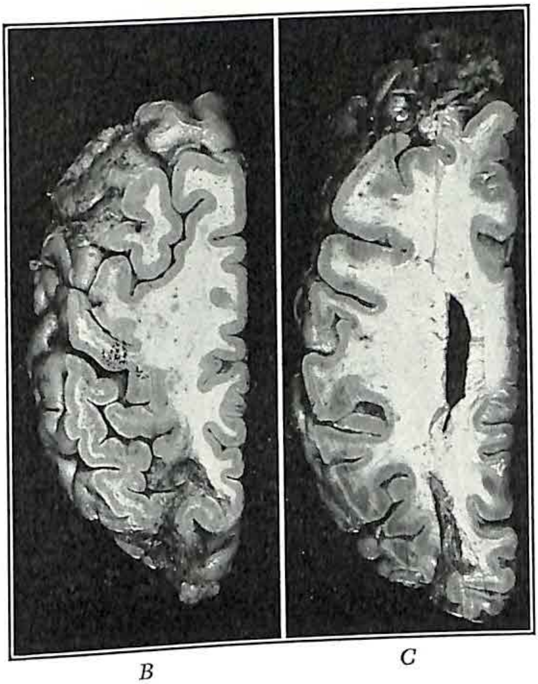
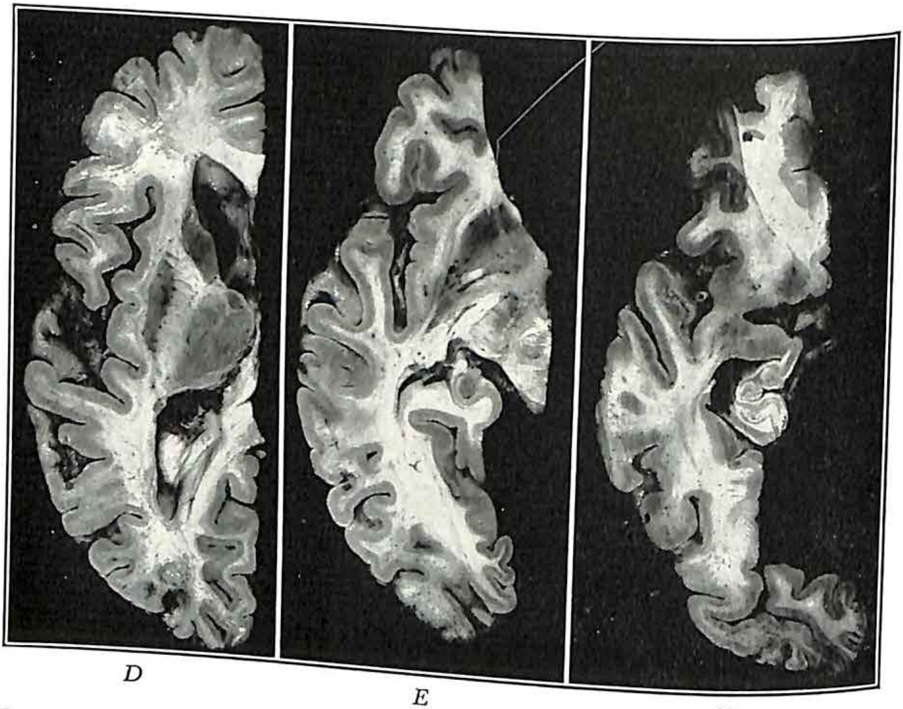


FIGURE 5. (*B*) Uppermost section, showing the softening in the left parieto-occipital region. (*C*) Second section, 14 mm below *B*.

Figure 6*A* and *B* is of a case in which the right half of the brain is the one involved. One can see a complete destruction of the posterior part of the occipital lobe. This was the case of a woman who had a vascular lesion at home many months before. She had acted "crazy" or "hysterical." Her sister-in-law was not at all sympathetic with her and told the husband, "Your wife must be crazy. She claims she can't see, yet she walks around objects and doesn't run into anything." She couldn't recognize her husband or any part of her own body and couldn't find her way to the bathroom. They would leave her out in the backyard, and when she asked to go to the bathroom, she was left there, as an experiment, but they had to clean up the mess. The husband then had her brought in and placed with her bed next to the bathroom door and said, "If you want to go, go yourself," and he and her sister-in-law sat and watched. After awhile, she said that she wanted to go to the bathroom, and when help was refused she soiled the bed. Because of this, she was sent to the psychopathic ward. They transferred her to the neurological ward, and here is her brain.



D *E* *F*

FIGURE 5. (*D*) Section through Broca's convolution. (*E*) Section showing cortical destruction of the mesial portion of lesion shown in *D*. (*F*) Section showing lateral portion of same lesion.

(*A, B, C, D, E, F* after J. M. Nielsen. *Agnosia, Apraxia, Aphasia*. New York: Paul B. Hoeber, 1946, 2nd Edition, pp. 237 and 238. By permission of the publishers.)

This was in the days before penicillin, and the patient died of bronchopneumonia. However, I'm grateful for the brain. In this case, both the animate and inanimate functions were out on that side.

She was a right-handed woman, but I can't say she was left-brained, because I can't be sure of that unless we get proof of it. She was right-handed, and the right occipital lobe went out. The amazing thing is that the left occipital lobe traveled all through life with the right occipital lobe exposed to the same visual impulses and the same training and experiences, yet when the right went out, the left was not able to do anything so far as associative functions were concerned.

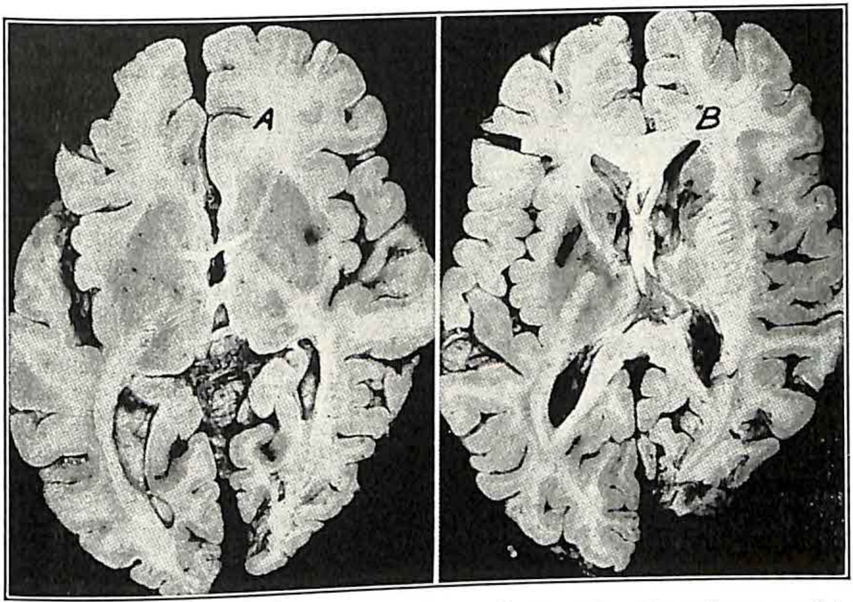


FIGURE 6. *A* is 1 cm. lower than *B*. The two horizontal sections show complete destruction of the right occipital lobe. (From J. M. Nielsen. *Agnosia, Apraxia, Aphasia*. New York: Paul B. Hoeber, 1946, 2nd Edition, p. 204. By permission of the publishers.)

. . . .

DR. MC CULLOCH: I would like, instead of discussing the papers, to throw this little thing into the general expression of ignorance that Dr. Klüver usually demands of us. About ninety years ago, it was discovered by Claude Bernard that sticking or pricking into the brain stem would cause a rise in the blood sugar, even spilling it into the urine. The mechanism for this control by the brain, of sugar in the blood, has been investigated many times. It has been shown that it can occur by (pathway number one) nerves to the adrenals, by (pathway number two) nerves to the Islands of Langerhans, in the pancreas, by (pathway number three) nerves to the pituitary, and thence, chemically. Dr. Leimdorfer injected into the cerebral spinal fluid quantities of adrenalin, beginning with very small doses and ending up with milligrams per kilogram. It never causes a rise in blood pressure, but it does cause an enormous rise in blood sugar which persists for many hours. So, instead of investigating the puncture of Claude Bernard, we investigated the effects of adrenalin in the cerebral spinal fluid. We tried to find the path from the brain. First, we removed the adrenals and then sectioned the vagi, or we took out the pituitary and sectioned the vagi. But no matter which paths we removed, the other apparently always sufficed. We were, I think, at that stage of the game, convinced that we had proof that the brain could use any one of the three ways. Unfortunately, we have now succeeded in removing all three, and it still works.

When we ask how our brain knows what our blood sugar level is, we are again in very much the same boat. You can fool your brain by holding, not necessarily sugar but anything that tastes sweet, under your tongue or around the edges of your tongue, and your brain will then pull down your blood sugar. You can fool it very beautifully in the cat by injecting saccharine. One of the ways that you use for measuring circulation time in man is to inject saccharine, and get a sweet taste. If you inject much saccharine, after that instantaneous sweetness, you can put all the sugar you want in your mouth and you can't taste it—you lose the sense of taste for sweets. When you do this to an animal, the brain at once regulates the blood sugar upward, as if there were too little sugar in the blood. As yet we have been unable to prove that sections of the nerves of taste will prevent the brain from regulating blood sugar. Therefore, each time we get one of these problems in which we are concerned on the one side with chemistry, and on the other side with the structure of the nervous system, we get into difficulties which, simple as the things may seem, take us years and years to solve.

DR. GERARD: I think that we should all be very grateful to Dr. Klüver for the framework that he has set up in his address. Certainly others are more competent than I to discuss the particular phenomena associated with these lobectomies. I was much impressed with his characterization of the occipital lobes as homeostating the external world. However, I am particularly grateful to him for, in a sense, putting the brain back in the body. He has touched upon one thing that has been very much in the air and yet has hardly come into the discussion;

the problem of psychosomatics. I wonder if the disturbances which resulted from his treatment of monkeys should be attributed to something like a severe look at the pituitary or even to mechanical manipulation of the brain, or if they are not simply examples, as he meant us to take them, of the various types of interactions between the brain and the rest of the body.

I was also very much pleased with the return to some chemical aspects which, though difficult to interpret, sometimes do come clean—even such crude overall phenomena as that of carbohydrate metabolism. Some work by a young pharmacologist is rather directly illustrative. He found that mescaline inhibited the oxygen consumption of brain, in vitro. He then tried a number of the typical substrates the brain can or does use, and found that succinate helped restore the oxygen consumption of the mescaline-inhibited brain. He then injected succinate into a number of students who had been given mescaline and were experiencing the rich visual hallucinatory complex so produced. The mescaline hallucinations were promptly wiped out by the succinate, and the subjects returned to normal. With insufficient succinate injected, the hallucinatory phenomena recurred after one-half to one hour and could again be eliminated by succinate injection. I think experiments of that sort do give us a little encouragement in this morass of uncertainty.

DR. LASHLEY: Dr. Klüver described the effects of removing the occipital lobe in the monkey as producing a response to the total flux of luminous intensity as contrasted with a condition where area and brightness are distinguished by the normal animal. We have found the same thing true in the rat. If in addition to destroying the striate cortex, the pretectile region and superior colliculi are damaged, the animal is rendered totally blind. These facts suggested a division of functions between the visual cortex and the tectum: the visual cortex specialized for reactions to the spatial distribution of excitations, the tectum for reactions to the intensity of excitation. Destruction of the tectum with the striate cortex intact did not, however, produce any visual symptoms. The discrimination of surface brightness and extent is unaffected. This illustrates a problem which is frequently encountered. The intact cortex seems to suppress the participation of lower centers in functions which they are capable of performing in its absence.

I agree with Dr. Nielsen's point that it is dangerous to generalize from one species to another. Removal of the prefrontal cortex in the rhesus monkey seems invariably to produce a definite syndrome centering around loss of the delayed reaction. We have now a number of chimpanzees lacking the prefrontal cortex. They show no such symptoms; in fact, we have not been able to discover any defects resulting from the operation. On the other hand, the agreement of experimental results with different species is much greater than the contradiction between them. When such contradictions do arise, they call for a searching and critical examination of techniques before their ascription to species differences is justified.

DR. WEISS: I think the reference to the fact that a great variety of different interferences can produce the same symptoms as do chemical agents is highly significant. It may signify that the symptom in question is a result of a chain of chemical reactions. It is easy to see that in such a chain the interference with any one step will preclude the terminal reaction or, in other words, produce the same deficiency symptom. We are reminded in this connection of the great multiplicity of mechanisms through which lethal genes exercise their effects. In such complicated chains it is unwarranted, of course, to infer that a variety of agents producing the same effect must have the same or similar points of attack or mechanisms of action. They may attack quite different parts of the process and in quite different ways.

DR. KÖHLER: I have perhaps missed the connection between two parts of Dr. Klüver's paper. He gave a fascinating description of the behavior of his monkeys, and he also discussed important pharmacological issues. At the present time, is there a bridge which leads from the former to the latter topic?

DR. KLÜVER: About twenty-five years ago I developed a method which I called the "method of equivalent and non-equivalent stimuli." And I have been wrestling with facts of equivalence and non-equivalence ever since. When Dr. Weiss referred to a multiplicity of agents producing the same result, he referred to facts of equivalence. The fact that a large number of heterogeneous stimuli produce the same behavioral effect implies the ineffectiveness of a large number of specific factors and stimulus variations. If you find widely different stimuli producing the same effect, it becomes necessary to locate the "identical" or "common" factor responsible for the equivalence. Unfortunately, the determination of such common factors is fraught with great difficulties. The experimenter will again and again find that he cannot draw generalizations at an early date when it comes to isolating the common factors underlying particular forms of equivalence. He may find a property or set of properties common to diverse equivalent stimuli; yet, on continuing experimentation, he may discover non-equivalent stimuli exhibiting the same common property. This means, then, that the particular factor he has isolated as being effective in producing a given reaction cannot be held responsible for the equivalence observed. It is the importance of non-equivalent stimuli which has frequently been overlooked in behavior analysis.

I wish to thank the discussers for their various remarks, but particularly Dr. Nielsen for adding such interesting data on agnosias. It is very likely that the temporal and occipital lobes of the monkey are in many ways different from those of man and, in fact, different from those of other mammals. It is of interest in this connection that Munk, who thought that he had succeeded in producing *Seelenblindheit* in dogs, failed to produce it in rhesus monkeys. It was subsequently shown by a number of investigators that the symptoms he produced in dogs were not due to a "psychic blindness," but to various visual disturbances and field defects. As regards the picture of psychic blindness in the bilateral temporal monkey, it seems to be less complicated by other factors than the visual agnosias in human patients. In considering clinical observations, I am impressed with the factor of selectivity operative in the agnosias. In man, disturbances of recognition are usually confined to certain types of objects and to certain kinds of relations between objects. One of Grünbaum's patients, for instance, had no difficulty in recognizing spatial relations except when his own body was the point of reference. Dr. Nielsen has provided some beautiful examples of such selectivity. Furthermore, a patient may be able to recognize certain details, objects, or relations one day but not the next or only under certain conditions, or he may recognize certain objects or properties spontaneously, but not by concentrated efforts. In contrast to this variability in human patients, the picture observed in the bilateral temporal monkey is really a monotonous one. The monkey tends to respond to every available object, no matter what its character, and constantly behaves as if unable to recognize the meaning of any object on the basis of visual criteria alone.

I wish to thank Dr. McCulloch for calling attention to the complexity of relations between structural and chemical factors. He has touched upon very important and exceedingly difficult problems. I was greatly interested in Dr. Gerard's statement that succinate injections suppress a mescaline-induced hallucinosis or at least decrease the complexity of mescaline hallucinations. I am curious to know whether sodium succinate will also be effective in banishing the hallucinations produced by *d*-lysergic acid diethylamide. Perhaps Dr. Alles, who is present today and who for many years has worked on mescaline and related compounds, is able to throw some light on this question. I believe that Dr. Alles' data on the oxidation of various amines with amine oxidase are of interest particularly in regard to mescaline.

To return to Dr. Lashley's remarks on the visual system, there is no doubt that further work is necessary to clear up species differences, particularly with reference to the role of the superior colliculi in mediating visual functions. I should like to add only one point to bring home to you what it means that the bilateral occipital monkey responds in terms of quantities of light. If a normal monkey, after having been trained to respond differentially to two luminous objects differing in intensity, is suddenly confronted with one luminous object exhibiting an intensity gradient, such as light transmitted through a neutral tint optical wedge, it ceases to respond differentially. In the case of the bilateral occipital monkey, however, there is no disturbance of the differential response. In making discriminations, the bilateral occipital monkey apparently relies on differences in the quantity of light received while occupying different positions in space with reference to the wedge. Similarly, it relies on differences in the quantity of light received in different positions when responding to two luminous objects occupying different positions in space. For a normal monkey, *two* luminous objects differing in intensity are not equivalent to *one* luminous object with an intensity gradient. This means, then, that the bilateral occipital monkey can perform "generalizations" which the normal monkey cannot perform. The normal monkey cannot "generalize" in terms of quantities of light since it responds to a visual world characterized by constancy phenomena and structured in terms of objects. Dr. Köhler asked me about the relation of chemical to behavioral studies. In analyzing behavior alterations following brain lesions, I was gradually forced to consider processes and events in the extracerebral environment. For instance, I could not ignore the role of the endocrines. There are indications that many relations between brain and behavior mechanisms cannot be understood without taking into account the interrelations of structural and biochemical factors.

In closing my discussion, I should perhaps not leave you with the pessimistic statement I made when quoting Clark's views on the laws involved in the reactions between drugs and living cells. Perhaps I was wrong in taking the stand that the laws of psychology, for centuries to come, will not be very simple affairs. Perhaps one should adopt a truly optimistic, American attitude in this matter. Perhaps one should approach the future in the spirit of the man who became world-famous for his treatment of neurasthenia and hysteria and who was also the first American to crystallize hemoglobin and even tried to distinguish males and females on the basis of hemoglobin crystals. Although he refrained from considering the relations of psychology to "blood, sweat, and tears"—he lived during the days of the Civil War—he did write a treatise on the relations of behavior to "fat and blood." I have, of course, reference to S. Weir Mitchell.

Relational Determination in Perception

WOLFGANG KÖHLER
Department of Psychology,
Swarthmore College

It is the main purpose of this paper to discuss records of electric currents which have been taken from the heads of human subjects under conditions of pattern vision. We have recently turned to work in this field, because it seemed natural to do so in view of certain psychological observations. I will therefore first describe these psychological facts.

When visual objects, figures, dots, or lines have for some time occupied a given region of the field, other visual objects which are now shown in this region tend to be displaced or distorted. Phenomena of this kind are known as "figural after-effects." The objects which establish such effects are called "inspection (or I-) objects," and the objects which exhibit the effects, "test (or T-) objects" (13).

According to the definition of figural after-effects which has just been given, a T-object is affected only if it is located within or near the area in which the I-object has been shown. This area is defined in a retinal (or cortical) sense rather than in terms of "absolute" space. Suppose, for instance, that after the I-period the eyes of the subject turn to a new fixation mark. Under these conditions, an after-effect will be observed in the new direction if the retinal place of the T-object has the right relation to the retinal area previously occupied by the I-object. The T-object need not differ from the I-object. When inspected for some time, many visual objects change their own spatial characteristics. In a way, they then serve both as I- and as T-objects.

Most figural after-effects can be derived from the following principle: T-objects recede from areas in which I-objects have been shown, particularly from the regions in which the contours of these objects have been located. As a result, T-objects may be simply displaced. Figure 1 shows a combination of I- and T-objects in which this is the case. (Actually, the I-object alone is presented first, and is then replaced by the T-objects. The simultaneous presentation of I- and T-objects in Figure 1 merely serves to show their spatial relation. The cross between the T-objects indicates the position of the fixation mark.) When, after an I-period of, say, one minute, the T-objects are presented, the one on the left side appears lower than its partner. The fact that the right T-object is not at all, or clearly less, affected demonstrates the localization of the effect.

Obviously, the left T-object may be shown above as well as below the area of the I-object; a T-object may also be shown on the left or on the right side of this area. In each case, it would appear displaced away from the I-object, if the second T-object were shown in a position which makes the displacement clear by comparison. Now it seems plausible to assume that inspection of an I-object alters the neural medium in which the process of this object occurs, and that a T-object is displaced because its process is affected by that alteration of the medium. But while inspection of a given object can cause only one pattern of alteration in the neural medium, we have just seen that this alteration may cause a number of different displacements, depending upon the location of the T-object in relation to the I-object. Consequently, we have to distinguish between the alteration of the medium as such and the various after-effects which may afterwards be demonstrated. These facts ought also to have different names. In previous reports the alteration of the medium has been called "satiation," and the term "figural after-effects" has been reserved for the changes which T-objects suffer when placed within or near satiated areas. These names will be used in the following discussion.

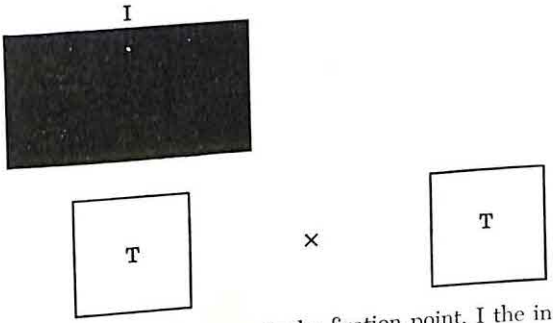


FIGURE 1. Typical stimulus figure. × is the fixation point, I the inspection figure, and T and T the test figures. (I is withdrawn before T is presented.)

It will be seen immediately that, with certain spatial relations between I- and T-objects, the displacement of the latter must result in changes of distances and sizes. In Figure 2, for instance, which again shows I- and T-objects in one pattern, both the T-objects on the right side must recede from the I-areas in their immediate neighborhood, and thus their distance will be shortened. At the same time, both the T-objects on the left side must recede from the I-object between them, so that their distance grows. As a consequence, the four T-objects together will appear to form a trapezoid rather than a rectangle. The size of figural after-effects in general varies strongly from one individual to another; but most observers find the present example particularly convincing.[*]

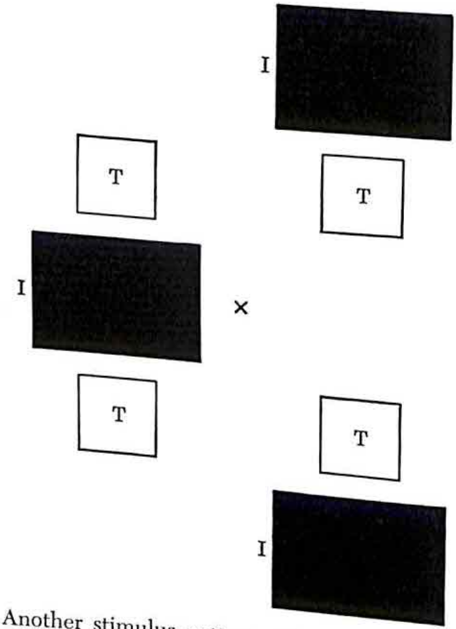


FIGURE 2. Another stimulus pattern. (See Figure 1 for explanation.)

While in the instance of Figure 2 it is empty distances which shrink or grow, in the following example (Figure 3) the size of an object is affected. After the I-period, the various parts of the T-circle on the left side recede from the outline of the I-circle, which constitutes a region of maximal satiation. Hence this T-circle must now be visually smaller than the objectively equal comparison object on the right side. This is actually the case. The effect can be measured by varying the size of the comparison object until it appears equal to the affected T-object. The distortion tends to be fairly conspicuous. The affected T-circle may lose more than 20 per cent of its area when the I-circle has been inspected for 45 seconds.

---

[*] At this point it should be mentioned that those who wish to experiment in this field ought to give sufficient attention to the quantitative spatial relations between I- and T-patterns, and also to the size of the distance between these patterns and the observer (see Köhler and Wallach, [ref. 13], pp. 297–303).

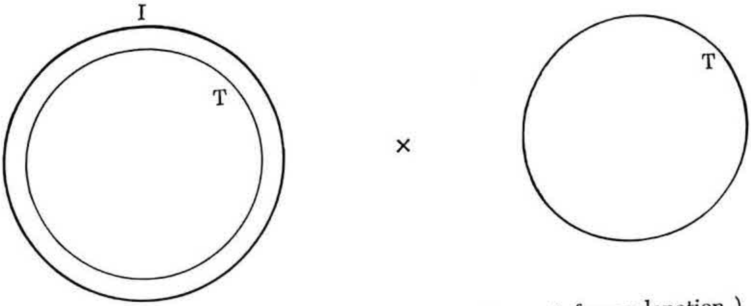


Figure 3. Another stimulus pattern. (See Figure 1 for explanation.)

The displacement of T-objects in the neighborhood of satiated regions follows a rule which may at first appear to be paradoxical. Generally speaking, the displacement is maximal, not in the region of maximal satiation, that is, when the T-object coincides with the I-object, but rather when the T-object is shown at a certain distance. Actually, this rule follows from the fact that T-objects recede from more into less satiated areas. Suppose that both the I- and the T-object are straight lines, and that these lines are parallel. Many observations prove that the I-object satiates not only the place in which it is located but to a degree also adjacent regions. If, however, the I-object itself is bilaterally symmetrical, and if the medium can be regarded as homogeneous, the resulting pattern of satiation must be approximately symmetrical about the same axis. This applies, of course, to the case in which the I-object is a straight line. As a consequence, there are two positions of the T-line in which it cannot be displaced. First, it cannot be displaced when shown at a very great distance. For here its process must be virtually independent of changes in the medium, which the remote I-object has established. Second, there can be no displacement when the T-object coincides with the I-object. For, in this position, the T-line lies between two areas in which satiation has approximately the same pattern and the same intensity so that the object cannot move for reasons of symmetry.* It follows from this reasoning that major displacements of the T-line are possible only when this line is shown at some distance from the I-line, where satiation is less extended and less intense on one side of the T-line than it is on the other side. Naturally, displacements must again decrease when, at still greater distances, the T-line entirely emerges from the satiated area.

Measurements of the displacement at varying distances from the I-line confirm this deduction. But there are further facts which can be derived from our argument. For instance, the T-line need not be parallel to the I-line. If the I- and the T-line form a small angle (see Figure 4),† one end of the T-line coincides with a point of the I-line, and can therefore not be displaced; but, from here towards its free end, its various parts have increasingly asymmetrical positions within the pattern of satiation, and must therefore be displaced by gradually growing amounts. As a result, the T-line must turn in space, away from the I-line. This is what actually occurs when the experiment is made. For the same reason, if the I-line consists of two straight lines which form an obtuse angle, the two halves of a straight T-line, which passes through the apex of this angle, must turn away from the two parts of the I-figure; in other words, the T-line must become a line bent in the direction opposite to that of the I-figure. That this really happens was first discovered by Verhoeff (17), and once more discovered by Gibson (5). Gibson also found that if an I- and a T-line intersect at a small angle, the T-line turns away from the I-line. Gibson, it is true, gave his excellent observations a different interpretation; but it can now no longer be doubted that these observations constitute figural aftereffects in the sense in which Wallach and I are using this term. The

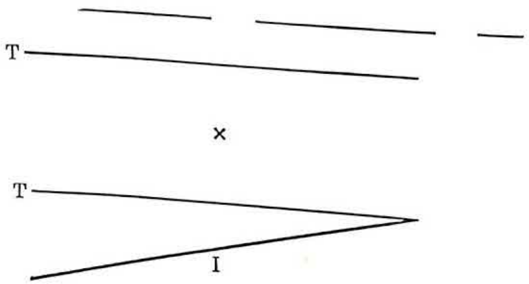


FIGURE 4. Another stimulus pattern. (See Figure 1 for explanation.)

---

* For physicists I will add that under these circumstances the T-line is in a position of stable equilibrium.

† The second (parallel) T-line is added as a neutral comparison object.

same holds for Gibson's further discovery that, after prolonged inspection of a slightly curved line, a straight line in the same location and orientation appears curved in the opposite direction.

By choosing and combining I- and T-objects in one way or another, we can readily produce a great many further after-effects. No more examples will be needed for our present purpose. I prefer to formulate some statements which refer to the nature of such effects in general.

1. Inspection of *any* visual objects gives rise to figural after-effects, provided that the objects are clearly segregated from their ground, and that the T-objects are shown in the right places.

2. It is the prolonged occurrence of the process of an I-object which, as such, establishes satiation and, if a T-object is afterwards shown, a figural after-effect.*

3. The locus of satiation is mainly cortical rather than retinal. For, after inspection of an I-object with one eye, a T-object seen with the corresponding region of the other eye also exhibits the after-effect.

In a more recent investigation, figural after-effects have served to answer an old theoretical question. It has been demonstrated that T-objects recede from the places of I-objects not only in the two dimensions of the frontal plane but also in the third dimension of visual space (10). In fact, all figural after-effects which are known to occur in the former case can also be observed in the dimension of depth. Moreover, the rules which they follow in the third dimension are identical with those which hold for the effects in the frontal plane. This applies also to the factor of localization. For instance, if with a given fixation mark a T-object is shown behind the place in which the I-object has been located, this T-object recedes to a greater distance; and if it is shown in front of that place it recedes from it toward the observer. (In both cases, the displacement is again greater at a certain distance from the place of the I-object than it is in its immediate neighborhood.) However, no effect is observed if, in retinal or cortical terms, the I- and the T-object lie in different directions. Such facts seem incompatible with theories, according to which the third dimension of visual space is merely an artifact for which processes of learning are responsible. It will be remembered that, some time ago, Lashley and Russell reported findings of an entirely different kind which also contradict such theories (14).

---

* A few remarks ought to be added to this statement. I do not wish to exclude the possibility that extra-visual factors influence the speed with which satiation develops. In fact, we have good evidence for assuming that, when attention is concentrated on an object, satiation in the area of this object is accelerated. Again, in referring to the prolonged occurrence of the process of an object, the statement does not imply more than that satiation grows with continued inspection. By using a technique of summation, Wallach and I have been able to show that low degrees of satiation develop when objects are shown only for fractions of a second. We are inclined to infer that virtually all perception of objects goes with a measure of satiation.

There is only one difference between the figural after-effects in the frontal plane and those in the third dimension. For the most part, the latter tend to be more conspicuous than the former. We have never found a subject who failed to be impressed by the following observation. On a piece of white cardboard a number of thick black parallel lines are drawn in a vertical direction, and then the surface is given a curved shape, convex toward the observer, who fixates a mark in the middle of the pattern. When after a while this surface is replaced by a plane surface, with black lines and a fixation mark in strictly corresponding positions, this T-surface appears strikingly curved in the opposite sense. With an I-surface concave toward the observer, the T-plane assumes an equally pronounced convex appearance. At first, such observations may seem to be open to criticism because on a curved solid surface there will always be gradients of illumination which must give rise to common negative after-images. It might be argued that only the appearance of such negative after-images on the plane T-surface causes the impression of an opposite curvature. Actually, the same effect can be demonstrated when gradients of illumination are entirely absent. For this purpose, we have merely to prepare two plane but slightly disparate drawings which, when fused in a stereoscope or haploscope, again give a surface curved in the third dimension. It will be found that after inspection of such a surface a corresponding pattern in the plane appears curved in the opposite sense. Since in this experiment both drawings are objectively plane, it is easy to give them a uniform illumination, and thus to exclude any explanation in terms of gradients of illumination and their after-images.

Vision is not the only sense modality in which satiation and figural after-effects occur. When Gibson had discovered that after inspection of visual curves straight lines tangent to these curves were seen as curved in the opposite direction, he immediately made a similar experiment in kinesthesis. Blindfolded subjects were asked to move the fingers of one hand along a curved edge, and to repeat the movement a great many times. When afterwards the same hand followed a straight edge, this edge appeared curved in the opposite sense. It seems hardly plausible to assume that the nature of this phenomenon is essentially different from the curved-line effect in vision.

We have observed and measured another after-effect in kinesthesis (9). In vision, the distance between two parallel T-lines is increased when the subject has previously inspected a narrow rectangle which lies between them. On the other hand, if the same T-lines are shown inside the area of a wider I-rectangle, they recede from its edges, and their distance is decreased. Can the same effects be obtained in kinesthesis? We asked our subjects to move the fingers of one hand along the two edges of a long rectangle with the width 0.5 inch and the fingers of the other hand along the edges of a rectangle with the width 2.5 inches. The movement was repeated back and forth for half a minute. Afterwards the subjects were asked to move their fingers along the edges of two equal rectangles, the width of which was 1.5 inches. We expected that, if there is satiation in kinesthesis, and if it has the same effects here as it has in vision, the T-objects would not appear to have the same width. Rather, since the width of one would be increased, and that of the other decreased, the former would appear wider than the latter. Results confirmed this expectation. Moreover, if one of the T-objects was replaced by a scale, that is, an object the width of which varied by small and equal steps, our subjects were able to find a width on the scale which appeared equal to the width 1.5 inches felt with the other hand. Although individual differences as to the size of the phenomenon are again great, we have so far found no subject in whom the effect cannot be demonstrated. Our arrangement often gives very large distortions. For fifty college students we found an average of 43 per cent. After a satiation period of 30 seconds, the width 1.5 inches, presented on the side of the narrower satiation object, appeared equal to 2.15 inches on the scale which was given on the side of the wider satiation object.

In preliminary experiments we found that similar facts occur when objects are felt by passive touch rather than kinesthetically. On the whole, it now seems possible that any process which has a spatial structure is capable of satiating the medium in which it spreads. As a matter of fact, there is no reason why satiation should occur only in perception.

If the processes underlying object perception always establish satiation, any physiological theory of these processes must be a theory of satiation at the same time. It follows that in making assumptions about the facts which underlie object perception we must exclude such processes as cannot be responsible for satiation in our sense.

Present thinking about the functions of the human cortex is based on discoveries which were made in the early years of this century. According to these discoveries, interrelations between the periphery and the cortex are, in both directions, mediated by short-lived impulses which travel along individual nerve fibers. It is customary to assume that cortical function as such is essentially of the same type; in other words, that impulses which arrive in the cortex merely start further impulses in next neurons, and so forth. This assumption is not fundamentally altered by the thesis that neurons may be arranged in circular chains, and that impulses may travel around such closed circuits for considerable periods. At any rate, most neurophysiologists seem to be convinced that the physiology of the whole nervous system can be written in terms of nerve impulses.

I doubt whether the behavior of figural after-effects is compatible with this view, because it seems that in our experiments it cannot be nerve impulses per se which cause satiation. This statement is based on the following fact. Suppose that during the I-period we show a narrow black rectangle on a white ground, and that afterwards we present two T-objects, one within the area of the black object, the other just outside, the short distance of both from the edge of the I-object being the same. Under these circumstances, it will be found that the T-object within the area of the black I-object is more strongly affected than the T-object in the white environment.* To be sure, this T-object is also affected, as can readily be shown by comparing it with a third T-object which lies at a great distance. But the essential point is that within the area of the black I-object satiation is clearly maximal. On the assumption that satiation is caused by nerve impulses it would seem to follow that the frequency and density of nerve impulses are higher within the black object than they are at any point in the surrounding white. Can this conclusion be reconciled with present views about the behavior of nerve impulses? Apparently, this is not the case. As a consequence, there appear to be only two ways in which satiation can be explained in physiological terms. Many will insist that the explanation be given in terms of nerve impulses. But this would imply a change in our mental picture of nerve impulses; for it has just been shown that the facts of satiation cannot be derived from the now prevailing picture. We shall later return to this point. As a second possibility, it may be assumed that nerve impulses as such are not responsible for satiation as investigated in our experiments. Then we must find another process by which it is actually caused. I propose now to discuss the latter alternative.

---

* With a solid black object it is advisable to use one eye during the I-period, and to observe with the other eye during the test. In this fashion, observation of the figural after-effect will not be disturbed by after-images in the usual sense. The same procedure may be used in the situations to which Figures 1 and 2 refer.

Obviously, if this alternative is to be seriously considered it must be applied to satiation in general. The way in which satiation develops within and around a white object on a black background does not essentially differ from that in which it develops within and around a black object on a white background. Satiation must therefore be given the same interpretation in both cases. Thus, on the premise that satiation in the region of a black object is not caused by nerve impulses, we shall have to assume that the same holds for satiation in the region of a white object.

By what other process could satiation be established? It appears that any speculation in this respect is restricted by conditions which are inherent in our experiments. According to our observations, *only* objects or patterns cause any demonstrable satiation. Hence, we must find a process which accompanies object or pattern vision rather than the perception of homogeneous surfaces. The assumption that such a process exists is supported by a fact which was first observed by Adrian (1). The alpha rhythm of the human brain is much more seriously disturbed by visual objects or patterns than it is by a bright homogeneous field. Adrian suggests that it is attention to which the alpha rhythm is so sensitive. But there remains the other possibility that, quite apart from this factor, the rhythm is strongly disturbed by a visual process which accompanies the perception of objects or patterns.*

From the point of view of stimulation, what is a visual object? It seems safe to say that, in terms of stimulation, an object is an area (or a volume) which differs from its environment either as a whole or along its boundary. We see things of any kind only when a relation of inequality obtains between the stimulation in one area and that in another, surrounding, area. Thus it seems plausible to assume that the process which goes with object or pattern vision is a relationally determined process, and that satiation is established in regions in which this process takes place for some time. Relational determination is not a familiar term. I will therefore add that relationally determined processes are extremely common in physics. For instance, if temperatures differ in two parts of a system, a current of heat energy is established which tends to equalize the temperatures. The direction of the flow depends upon the direction of the difference, and in the absence of any difference there is no flow. Similarly, if a solution which contains certain molecules is surrounded by a second solution which contains these molecules in a different concentration, a current of diffusion will be observed, unless the solutions are separated by an impermeable barrier. The current flows as long as the concentrations differ. Thus, it is again a relation of inequality between the two parts of the system which maintains the process. Incidentally, our examples exhibit relational determination in more than one sense. As the currents of heat or diffusion spread, their distribution in space depends upon the shape of the boundary at which the parts of the systems are in contact. This shape is defined in terms of geometrical relations among parts rather than of merely local conditions, and the distribution of the flow adapts itself to such relations. Therefore, not only the flow as such is relationally determined, but the same holds also for its pattern in space.*

---

* It is, of course, well known that the alpha rhythm may also be blocked by non-visual factors. I am here ignoring such evidence merely because it is not related to the present discussion.

The examples given are by no means the only examples of relationally determined processes in physics. I need not mention further instances. In the present connection, it seems more important to remark that some such processes cause obstructions in the medium in which they occur, and that in this fashion after-effects are established when later further processes spread in the same medium.

Are any relationally determined processes likely to occur in the brain when objects or patterns are seen? Ten years ago, I suggested that the relationally determined process which underlies pattern vision is a direct electric current, and that such a current flows when conditions of excitation in one part of the visual cortex differ from those in an adjacent part.† An attempt was also made to explain how the electromotive forces originate which drive the current from one part to the other, and back again to the former. The explanation involved no hypothesis which is at odds with available knowledge of nerve impulses and their influence upon cortical tissue. Rather, those forces were derived from concepts which play a great role in present neurophysiological discussions. Nevertheless, this particular part of the theory need not now be described, because there may be various ways of deriving electromotive forces which would drive a direct current through the tissue. It seems that, whatever our choice may be, the distribution of the flow as such would always be about the same. It is this flow which we will now consider.

---

* Relational determination of events must be distinguished from certain other facts. Not all instances in which two or more factors participate in causing an effect are, for this reason alone, instances of relational determination. For example, in synaptic transmission many cells are thrown into action only if more than one impulse arrives at their surfaces. A combination of several causes in this sense need not, of course, involve relational determination. In other words, to be relationally determined by two conditions is one thing, and summation of the effects of mutually independent causes is another thing.

† W. Köhler (7), pp. 212–215, 239–244, 351, and 370 f. See also W. Köhler (8), pp. 55–106. A more detailed account is given by W. Köhler and H. Wallach (13), pp. 322 ff.

The flow would spread through the tissue as a volume conductor, which is to say that, in this connection, the brain must be regarded as a continuous medium to which principles of continuity physics apply.* In this respect, I merely emphasize a possibility which is implicit in present neurophysiology, even though its consequences have, until recently, not been explicitly considered. Surely, if the potentials of the alpha rhythm as well as those of "on" and "off" effects can spread through the skull, there is nothing *in* the brain to prevent such potentials from spreading through this medium as a continuum. As a result, it can hardly be a disturbing thesis that a steadier flow would do the same. In fact, the possibility that the brain is pervaded by field potentials was discussed by Gerard before I formulated the present theory (2). Later, he and Libet pointed out that functional interrelations in the frog's brain can best be interpreted from this point of view (3, 4, 15). Although the authors did not consider problems of perception, their views and mine may prove to be different versions of essentially the same conception.

In flowing through a continuum, a direct current assumes a distribution which, just as in our previous examples, is relationally determined by the shape of given boundaries. In object or pattern vision, the boundaries in question would be those between cortical areas in which retinal stimulation establishes different kinds or degrees of excitation. Wallach and I have indicated how the resulting distribution of the flow could be related to facts of space perception.† But in our present connection this particular question need not be raised. It will suffice if we consider a fact which concerns only the distribution of the current as such, and is quite independent of further theorizing. If excitation within a circumscribed cortical area differs from that in its environment, the resulting current must circle around the boundary at which the two areas are in contact. Moreover, unless the surrounded area is very large, the current must be denser in this area than it is in the environment in which it can spread widely. This is true whether or not local excitation is higher in the circumscribed area. Thus, if a black object is shown on a white background, the density of the flow must be maximal within the area of the black object, just as it is maximal within the area of a white object surrounded by black.

---

* I am aware of the fact that physicists sometimes use the term "continuity physics" in a different sense, in which it is contrasted with "quantum physics."

† Reference 13, pp. 332 ff.

We can now turn to the problem of satiation. The present theory has no difficulty in solving this problem. Any direct currents which flow through the nervous system polarize the surfaces of cells, and also change their polarizability. Generally speaking, this effect, the so-called electrotonus, has the character of an obstruction. Further currents which afterwards flow through the same medium are weakened. At the same time, they suffer changes of their distribution in space. We are not introducing a special hypothesis if we assume that the currents of our theory are also electrotonically active, and that the resulting obstructions follow the same rules as hold for electrotonus in general. For instance, the degree to which the various parts of the medium are electrotonically affected is directly related to the density of the current in those parts. Now we know that the density of the currents postulated in our theory must be maximal within the area of a circumscribed object, still great in adjacent parts, and progressively lower at greater distances. Consequently, the intensity of the obstruction which the currents establish must exhibit the same distribution. But this is also the distribution of satiation as investigated in our experiments. Thus we are led to the conclusion that satiation is merely another expression for electrotonus.

A more specific consideration of figural after-effects tends to confirm this conclusion. For example, the fact that satiation is more intense within the area of a black I-object than it is in its white environment now offers no problem. For, since in this case also the current must flow with greatest density through the restricted area of the object, its electrotonic action will again be maximal in this area. Quite apart from this special condition, how are the distortions to be explained which T-objects suffer in satiated areas? Obviously, if there are currents which go with I-objects, there must also be currents corresponding to T-objects. But the current of a T-object will be weakened and distorted, if the current of an I-object has previously pervaded the same region, and if its electrotonic effect still persists. To a degree, we can tell how the obstructions established by the currents of given I-objects must distort the currents of T-objects; and, if certain general premises are accepted, we can also compare such distortions with actually observed after-effects. It seems at present that biophysical theory and psychological observation are in satisfactory agreement.

Our interpretation of figural after-effects rests upon the assumption that the processes underlying object vision are direct currents. This assumption as such is more important than the fact that it seems to give an explanation of those particular phenomena. Under these circumstances, a direct test of the main thesis appears desirable; and, since the thesis has a strictly physiological meaning, it can be directly tested only in physiological experiments. Recently, Mr. Richard Held and I have done some experiments of this kind. I am grateful for grants from the American Philosophical Society, the Viking Fund, and Swarthmore College, which have made our investigation possible.

In dealing with our problem, physiologists would probably prefer to take records of electrical activities from the visual cortex itself, and therefore to work with animals. The advantages of this method are obvious; it will undoubtedly be used in future experiments. It seemed to us, however, that recording from the intact heads of human subjects would be particularly convenient in a first exploration. We realized, of course, that with this procedure negative results would not be decisive; for, even if pattern vision were associated with direct currents in the visual cortex, differences of potential on the surface of the head might be too small to give clearly discernible responses. But we hoped that the size of the potentials would be comparable to that of alpha potentials. If this assumption proved to be correct, our procedure was obviously justified.

As an amplifying system, we used the d-c amplifier which has recently been constructed by engineers of General Motors. This excellent instrument avoids the disadvantages of direct-coupled amplifiers, and yet strongly amplifies weak direct currents of any duration. I need not here describe the construction of this instrument since a good description can be found elsewhere (16). Records of the output were taken with the well-known photoelectric recorder of the General Electric Company. The subject was comfortably seated in an armchair to which an adjustable headrest was attached. He was surrounded by a shielding cage. We used electrodes of the silver-silver chloride type, inserted in glass tubes. Contact with the scalp was made by means of a saline solution (10 per cent) which filled the tubes. One electrode was placed about 1 centimeter above the occipital protuberance, slightly to the left of the median plane. Its precise position varied by small amounts from one experimental session to another. The second electrode was attached to the vertex. This electrode was connected with the (grounded) second lead to the amplifier and with the shielding cage. Any steady potentials which appeared in the absence of visual stimulation were compensated for by a balancing circuit.

In a first attempt of this kind we cannot expect to obtain records which are as elegant as those of nerve impulses registered with modern techniques. Compensation for potentials extraneous to the present problem is possible only so long as such potentials do not vary while records are taken. But if the duration of a record is long, gradual drifts of the base line and also more short-lived fluctuations do occur. While continued experience has gradually taught us to keep such disturbances within narrow bounds, their influence is still obvious in the records which I will now discuss.*

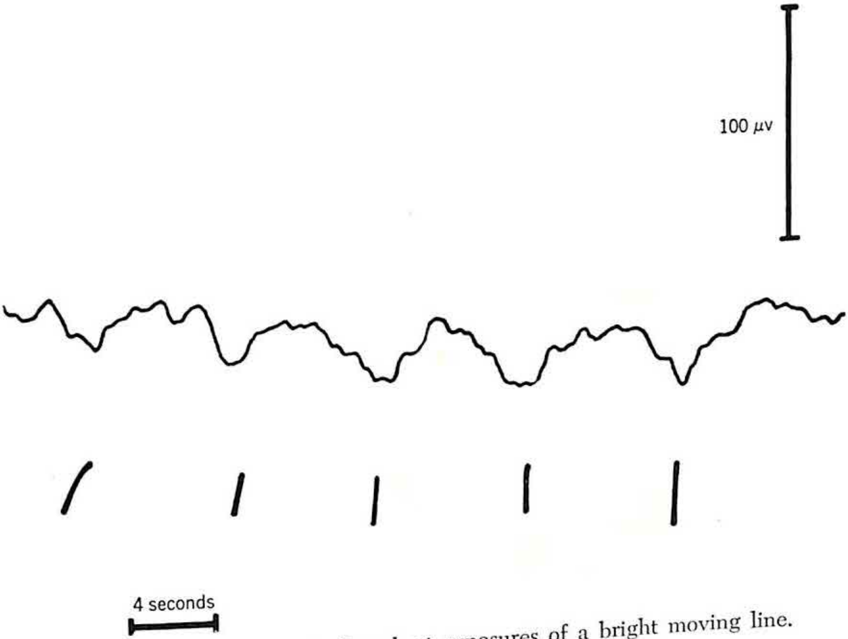
For two reasons, we worked with slowly moving rather than with strictly stationary visual objects. In the first place, it seemed possible that electric responses would be sufficiently large only if the occipital electrode had just the right position with regard to the cortical representation of the object. We did not, of course, know precisely what this position was when the object occupied a certain region of the visual field, and we did not wish to rely on mere trial and error in this respect. The difficulty could easily be avoided by moving a narrow bright rectangle of considerable length at right angles to this length across a faint fixation mark. A moving object of this kind had sooner or later to pass through the optimal position, if there was such a position. Our second reason for using this procedure was the fact that any direct currents which might spread in the visual cortex would tend to polarize the tissue, and thus to reduce their own intensity. We assumed that this effect would be weakened if the object were moved across the field; because under these circumstances the most intense flow, the current through the area of the object, would in no position have time to cause seriously disturbing electrotonus. Actually, we seldom used velocities higher than about 4 degrees of visual angle per second.†

Records I, II, III, and IV refer to the following situation. A bright horizontal bar moves across the field in a vertical direction. The field is not completely dark, but faintly illuminated by a weak

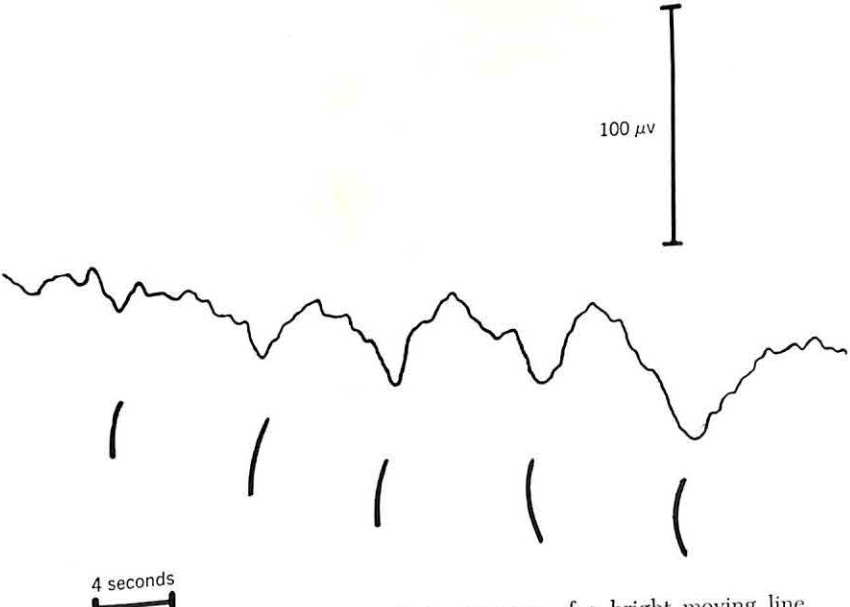
---

* These records were taken early in 1948. More recent records will be found in a paper by R. Held and the present author (12).

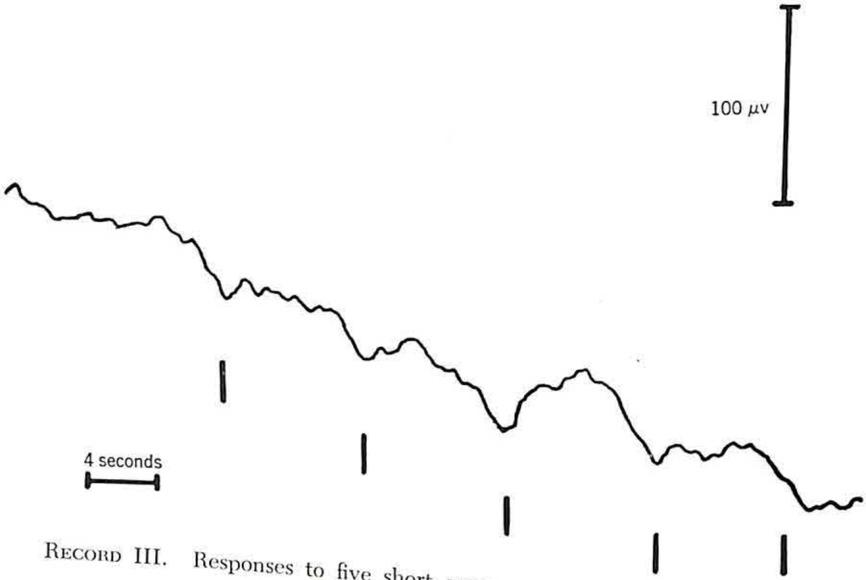
† More recent experiments have shown that it is by no means impossible to obtain responses when the object is stationary. But it seems true that such responses are more difficult to obtain; and for first experiments in this field we still recommend the use of slowly moving objects.



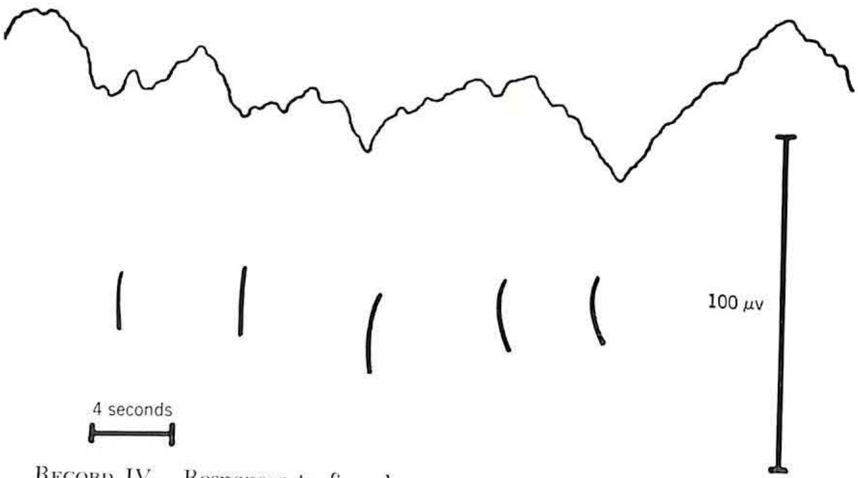
Record I. Responses to five short exposures of a bright moving line.



Record II. Responses to five short exposures of a bright moving line.



Record III. Responses to five short exposures of a bright moving line.



Record IV. Responses to five short exposures of a bright moving line.

lamp, the light of which allows the experimenter to deal with the instruments.[*] There are no contours in the neighborhood of the moving object. This object appears moving above or below the fixation mark, moves toward this mark, passes it, and then disappears on the other side. After a few seconds, this movement is repeated, and so forth, until the bar has crossed the field five times in succession. The extension of each movement amounts to about 15 degrees of visual angle.[†]

The records clearly show responses to the bright moving object. In nineteen out of twenty cases, deflections downward can be recognized where marks are given below the curves. These marks, made by hand at the time of the recording, indicate the moments at which the object actually passes the fixation point. It will be seen that the intensity of the responses tends to be maximal at these points.[‡] In examining the records, we have to keep in mind that they are written on very slowly moving paper. The distance of 1 centimeter corresponds to 4 seconds. Thus, while each response has the character of a wave, the duration of such a wave amounts to several seconds. That of a whole record with its five exposures is greater than half a minute.

While considerable drifts of the base line have not been able to obscure the responses, the difficulties inherent in such experiments ought not to be underrated. Sometimes "random" influences are obviously at work. In Record III, the last response is not particularly conspicuous, and in Record IV we can hardly discover the fourth reaction. The size of the deflections varies from a minute response (in Record IV) to excursions which correspond to about 50 microvolts (in Record II). All responses have the same direction. Comparison with a source of known polarity shows that while the bright object moves across the field the occipital surface potential is positive in relation to the vertex potential.

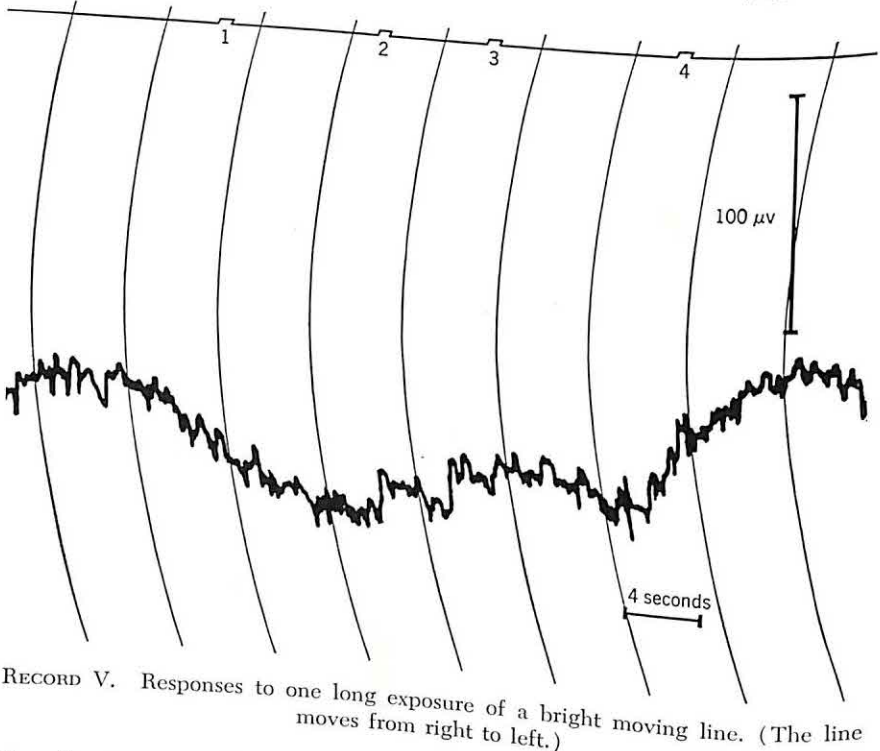
Records V, VI, and VII refer to a different visual situation. The horizontal bar is replaced by a vertical bar, and the movement across the fixation mark occurs in the horizontal rather than in the vertical direction. Moreover, the extension of the movement now amounts to about 120 degrees of visual angle, eight times the size of the movement in Records I to IV.° The latter change was made for the following reasons. *First,* in Records I to IV the form of the responses is that of relatively short-lived waves. Is this form caused by the fact that the duration of the exposures is short? Or are such responses short-lived under all circumstances? If the object moves across a wider area, while its retinal speed remains about the same, results may decide between the alternatives. *Second,* if the size of the responses actually grows as

---

[*] Since this was written, it has become obvious that extreme differences between the brightness of the object and the brightness of its environment must be avoided if good responses are to be obtained.

[†] In these first experiments, the distance from the eyes of the subject to the moving object was about 2.5 meters.

[‡] Owing to the slowness of the movement, the position of the marks is fairly accurate.



RECORD V. Responses to one long exposure of a bright moving line. (The line moves from right to left.)

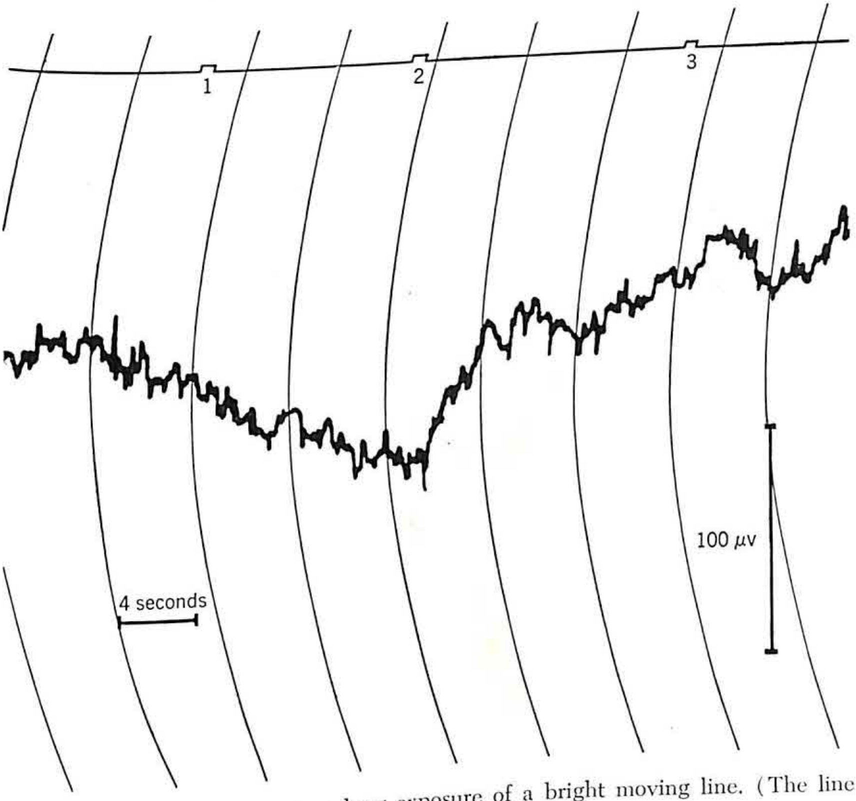
the object approaches the fixation mark, and as its cortical representation moves toward the foveal region of the visual cortex, Records I to IV may be interpreted in terms of cortical localization. For, although details remain unknown, the part of the visual cortex which lies under the occipital electrode must correspond to the foveal region of the visual field, including the fixation mark. From this point of view, it would not be astonishing if responses were maximal when the object passes this mark. But convincing proof of the fact would tend to show that the currents of our records really issue from the visual cortex. Otherwise,

---

° The widening of the movement in terms of visual angle was achieved partly by showing the moving object at a shorter distance from the subject.

why should their behavior agree with what is known about localization within this area? Our evidence in this respect would be greatly improved if, with a far more extended movement, the currents were to show the same behavior during longer periods of recording. The position of the active electrode must, of course, remain unchanged.

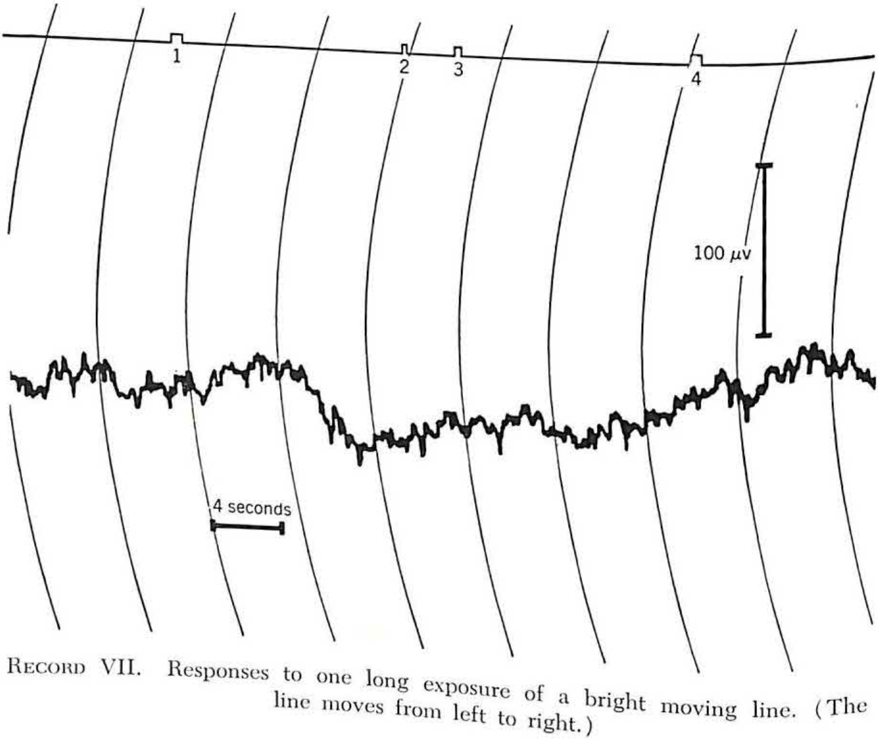
The vastly increased duration of a single movement makes it impossible to repeat exposures in a continuous record. In Records V to VII, it takes the object almost half a minute to cross the field. For one such period, we can generally have confidence in the process of recording; but drifts will go too far, and may also be reversed, if the time of recording is greatly increased beyond that limit.



Record VI. Responses to one long exposure of a bright moving line. (The line moves from right to left.)

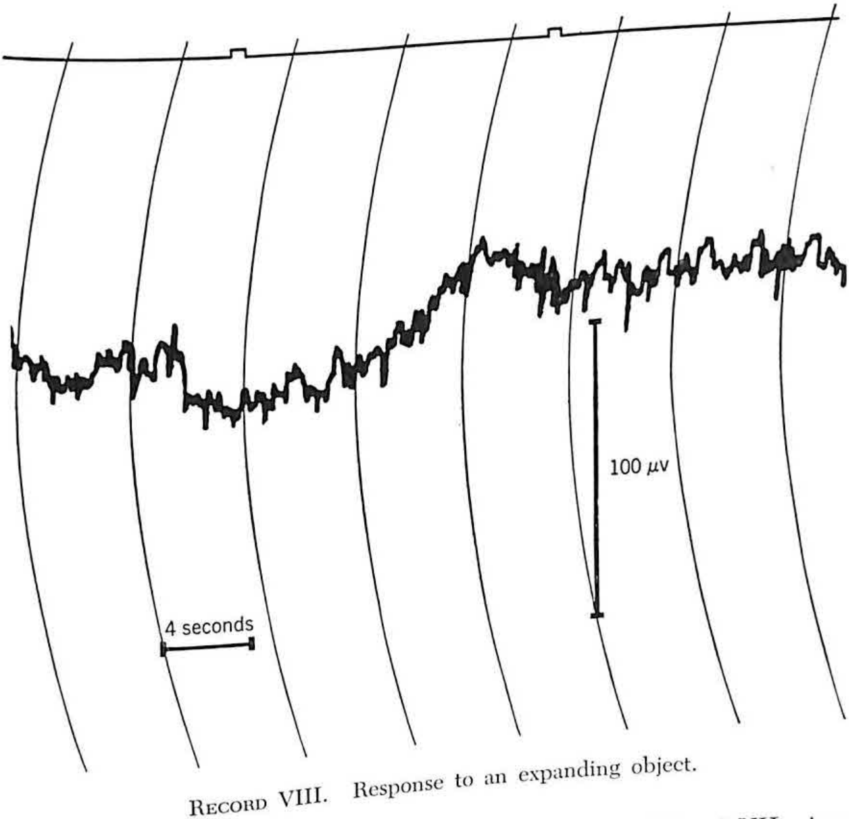
The paper speed of the recorder is the same in Records V to VII as in Records I to IV. But the galvanometer of this instrument is now replaced by an element with a lower response time (about 0.04 second). While in this fashion distortions of the shape of the responses are reduced, it may be regarded as a disadvantage that, after the change, rapid fluctuations are recorded which do not appear in Records I to IV. Since they swing and do not seriously obscure the recording of the steadier flow, no attempt has been made to eliminate them. After all, they may partly originate in the brain.

The signals above these curves indicate crucial positions of the moving object. They were made by an assistant who watched the object and closed the circuit of an electromagnetic marker when those positions were reached. The signals are slightly retarded; but the error cannot be greater than 0.3 second. In more recent experiments, we have also marked by means of photoelectric cells activated by the bright object itself, and with other automatic devices.



Record VII. Responses to one long exposure of a bright moving line. (The line moves from left to right.)

Records V to VII give a much clearer view of the currents under investigation than can be obtained from the preceding curves. In the first place, the length of the responses is now greatly increased, obviously because the object moves across a much wider field, and is seen correspondingly longer. In Record V, for instance, a deflection of constant polarity, and of the same direction as in Records I to IV, persists for 30 seconds, which is slightly more than the time during which the object is seen (compare the distance between the signals 1 and 4). In the other records, the responses are not much shorter. It follows that the responses in Records I to IV have been short-lived only because the exposures of the object have been short. Second, it is now quite obvious that when the object occupies foveal or parafoveal positions responses are particularly strong. (In Records V and VII, signal 2 refers to the moment at which the bright bar just arrives at the fixation mark, and signal 3 to the moment at which the object has just passed this mark. In Record VI, signal 2 indicates the position in which the fixation mark lies in the middle of the moving bar.) Outside this region, the flow through the leads decreases either gradually or more abruptly. This is, of course, the form which responses must exhibit if the currents of the records issue from the visual cortex, and if, while the object moves, they migrate first toward, and then again away from, the region under the occipital electrode.



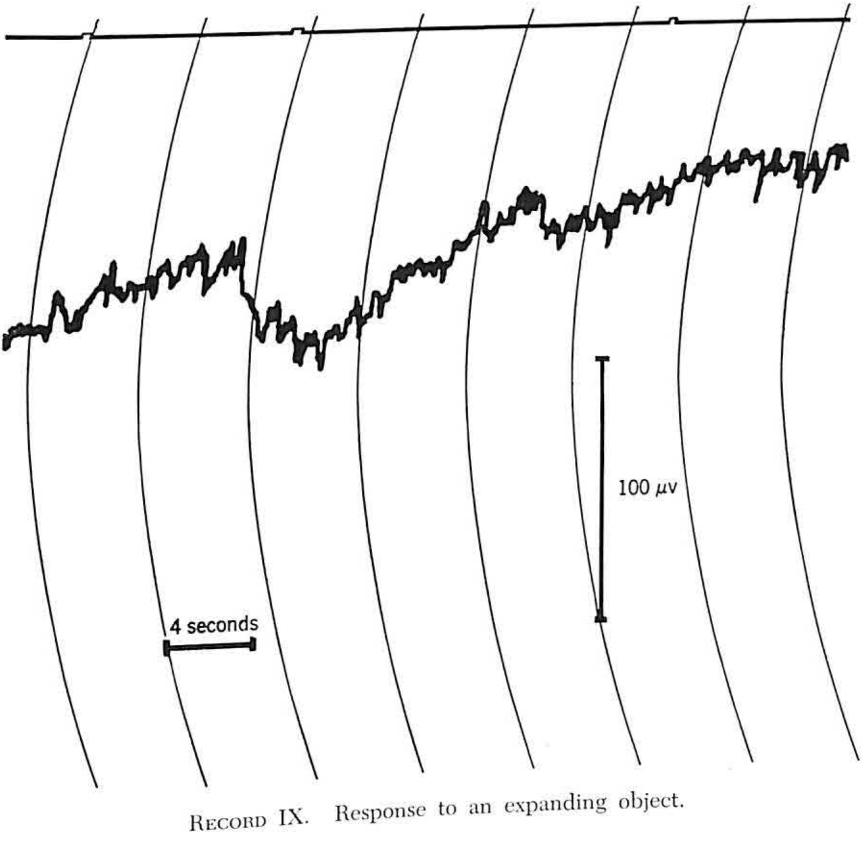
Record VIII. Response to an expanding object.

Actually, correlation with facts of localization within the visual cortex seems to go still farther. The responses are clearly not symmetrical with regard to the fixation mark. In Record V, the size of the deflection decreases by about 20 microvolts as soon as the object reaches the fixation mark and moves from one half of the visual field into the other. In Record VI, a much more striking decrease of the response occurs in the same region. In neurological terms, therefore, the intensity of the current which passes through the electrodes is greater or smaller, depending upon the hemisphere in which the cortical object moves at the time. Now, in Records V and VI the object moves from the right to the left periphery of the visual field, which means that its cortical counterpart moves first in the left and then in the right hemisphere. Thus the records show that deflections are greater in the left hemisphere. But this is the hemisphere in which they must be expected to be greater, if localization within the visual cortex has any influence upon the records. For both electrodes are placed on the left side of the median plane. Record VII corroborates this interpretation of Records V and VI. In Record VII, the object moves in the opposite direction, from the left toward the right periphery, and its cortical counterpart from the right into the left hemisphere. The electrodes occupy the same positions as before. It will be seen that while this record is also asymmetrical, its asymmetry has the opposite direction. There is hardly any response so long as the object moves through peripheral parts on the left side of the visual field. A sudden deflection occurs as the object approaches the fixation mark; and this response decreases only slowly as the object approaches the right periphery and its cortical counterpart moves in the left hemisphere. Again, therefore, a more intense flow passes through the electrodes when the cortical object moves in the hemisphere corresponding to their location.*

Records VIII, IX, X, and XI show that responses can also be obtained when the object does not move in the usual sense of the word. In these experiments, a small bright figure appears in the region of the fixation mark, and then expands to the right and to the left until a bright bar extends across the field. In all four records, an abrupt deflection occurs when the object is first shown, and begins to grow. This is once more a response which can readily be understood if the currents issue from the visual cortex. For, from this point of view, the cortical object now appears at once near the occipital electrode. Later, when the boundaries of the object move toward the periphery, while its center remains stationary, the size of the response decreases, more strikingly in Records VIII and IX than in Records X and XI. Such a decrease is to be expected if the responses actually represent object currents in the sense of our assumption. For, according to this assumption, the lines of flow which pass through skull and scalp, and then enter the "active" electrode, obviously issue from the middle of the cortical object. This part of the flow will be relatively strong so long as the object is narrow; but its intensity must be reduced when the object expands, and when at the same time the electrotonic obstruction gradually grows.

---

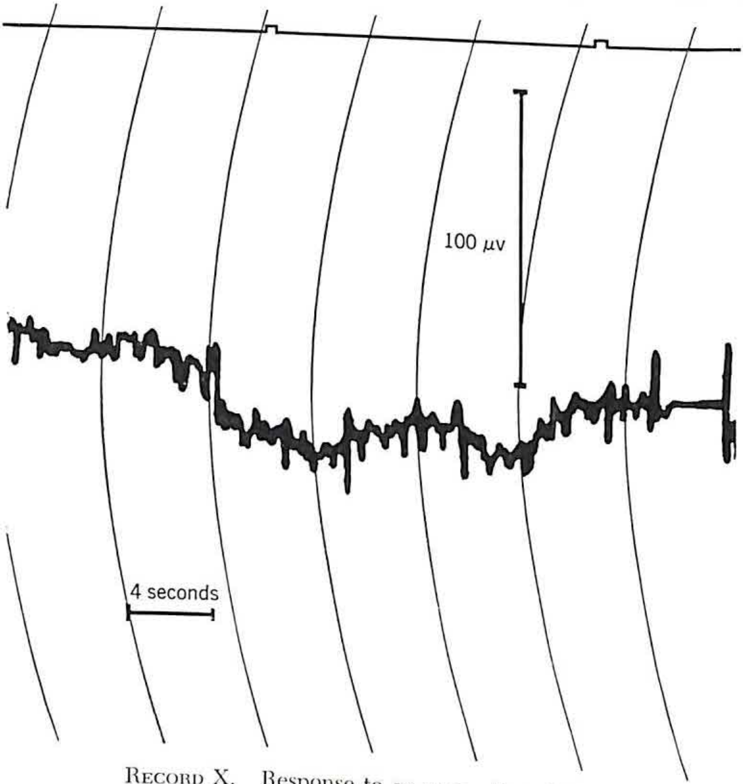
* Several records which are reproduced here show a phenomenon to which we did not give much attention when these early records were taken. Toward the end of the exposure or immediately after the object has disappeared, there tends to be a smaller second deflection which has the same polarity as the original response. Further experimentation has left no doubt that this phenomenon is a genuine after-potential. After-potentials are particularly obvious in Records V, VI, VII, and IX.



RECORD IX. Response to an expanding object.

The polarity of the currents is the same in these records as in the other curves. Potentials at maximal response vary between about 25 and 40 microvolts. Naturally, in these experiments the flow persists for less time because the exposures are shorter (note spacing of the signals).

We do not, of course, claim that, on the basis of these records alone, the thesis of direct pattern currents must be accepted. It will be the task of more than one laboratory to decide precisely what the records mean; and the number of possible interpretations can gradually be reduced only if, in the future, conditions of experimentation are far more widely varied.

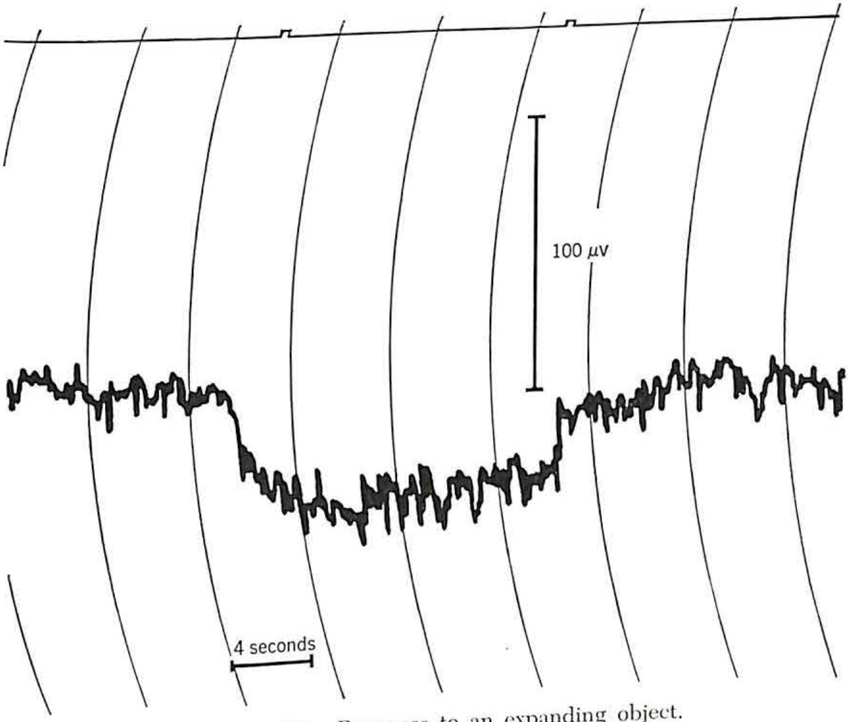


Record X. Response to an expanding object.

Apart from the assumption which has led to our experiments, what interpretations of the records ought to be considered?

1. Since the records were taken while contours moved across the visual field, it might be suggested that the subject's eyes followed these contours, and that, in some fashion or another, the responses were caused merely by such eye movements. I very much doubt whether this interpretation is tenable. Apart from the fact that a trained subject

is unlikely to make major eye movements when an object slowly moves across the fixation mark, the thesis is not compatible with records taken when eye movements actually occur. It is by no means difficult to obtain responses when the subject moves his eyes from one faint fixation mark to another. Nor is it necessary for this purpose that one electrode be placed near the eyes. Nevertheless, some eye movements give responses only if certain positions of the electrodes are avoided. For instance, when both electrodes are placed on the dorsal part of the head, quite near the median plane, and on the same side of this plane, any major movement of the eyes upward or downward is easily registered. But with the same position of the electrodes there are no responses when the eyes move horizontally. Now, this is precisely the position in which the electrodes were placed when Records I to XI were taken. Consequently, whenever our subject moved his eyes horizontally, this movement as such could not show in the record. Presumably, any eye movements which the subject made were in the direction in which the object moved, and then back to the fixation mark. Thus, while the present interpretation could perhaps be applied



Record XI. Response to an expanding object.

to Records I to IV, in which the object moved vertically, it is not acceptable in the case of Records V to XI, where we moved the object in the horizontal dimension.*

2. Is it possible to explain the responses as records of psychogalvanic reflexes? If this were the right explanation, visual stimulation would be only the remote cause of such responses, and the currents would issue from the scalp rather than from the visual cortex. We are inclined to regard this suggestion with considerable skepticism. In the first place, the latency time of psychogalvanic reflexes is known to be quite long, about 2 seconds in the case of visual stimulation. No delays of this size are observed in our experiments, if the object appears at once near the fixation mark, and its cortical counterpart near the occipital electrode. When using a higher paper speed and automatic marking, we find that under such circumstances responses always begin within a fraction of a second. Second, this interpretation is at odds with our records inasmuch as the size of the responses appears to depend on facts of localization within the visual cortex. For instance, if the currents do not originate in the visual cortex, why should responses tend to be stronger when the cortical object moves in the hemisphere corresponding to the place of the occipital electrode?

3. We have been asked whether the currents could not issue from the retinae. We hesitate to believe that they do. The spatial relations between the two retinae and the two projection areas of the visual cortex are by no means simple. There is no obvious reason why direct currents with sources in the eyes should spread to the projection areas in a way which agrees with those complicated relations. And yet, the responses appear to be related to the positions which the object occupies in the projection areas. I am also assured by experts that so long as the electrodes are attached to the dorsal part of the head they will hardly be affected by retinal currents.

4. As a last possibility, we must now ask ourselves whether our records can be explained in terms of nerve impulses which arrive in one part of the visual cortex after another while the bright object moves across the field. Among the rival interpretations we have to consider, this is probably the most important. Obviously, it can be defended only if the term "nerve impulses" is now understood in a broad sense. In referring to nerve impulses, we commonly think of

---

* Since this was written, responses quite analogous to those shown in Records I to IV have also been obtained when the object moved in the horizontal direction (12). The location of the electrodes was the same as in the experiments to which Records I to IV refer.

events which occur in fibers and in their most immediate environment. From these impulses we distinguish their fields which, we admit, spread farther. The distinction has a strong influence on our thinking. There are theories which deal with nervous function in terms of nerve impulses in the narrower sense only, and practically ignore their fields. But nerve impulses and their fields can hardly be treated as separate entities. If I understand the situation correctly, the nerve impulse is a current which originates when surface properties at one point of a fiber differ in a certain fashion from those at other points. This current flows partly in the fiber, but necessarily also through the surrounding tissue; and the latter part, the field of the impulse, belongs to this impulse as much as does the flow within the fiber.

The field pervades the tissue as a continuum. But it is also only the field which could have any effect upon electrodes attached to the scalp. Hence, if the responses in our records were caused by nerve impulses, the volume through which their fields spread would be enormous, because it would include both skull and scalp. As a matter of principle, this implication may not be particularly disturbing; but, if it were accepted, we should perhaps no longer say that impulses are propagated in fibers. The expression would be too seriously misleading.

More in particular, the thesis which we are now discussing might be given one form or another. It could be assumed either that the impulses which are supposed to cause the responses are synchronized, or that their temporal relations approximate a random distribution. In the former case, the thesis would be entirely compatible with our records as such. The instruments which we are using would not distinguish between a steady potential and a very rapid sequence of synchronized short-lived fields. At first, it seems therefore quite possible to maintain that the responses are caused by synchronized nerve impulses. Nevertheless, this explanation meets with a difficulty. I suspect that, if such synchronized impulse fields actually reached the surface of the head, their occurrence would already be a well-known fact. Ever since Berger's discovery, experimenters have often attached electrodes to the parts of the head at which our electrodes were located. Surely, many attempts have also been made to register electric oscillations which might appear when the subject sees a bright object. For this purpose, the amplifying systems must have been adapted to virtually all frequencies which have to be considered when impulses are being recorded. So far as I know, no persistent and well-defined oscillations have been found under these circumstances. And yet, if the impulses in question were synchronized, why should they not appear in records?* Voltages of more than 20 microvolts can readily be registered with any good amplifier. Thus it seems doubtful whether there are such synchronized impulses. I will admit, however, that further tests are desirable.

Can records such as ours be explained by the action of non-synchronized impulses? Since this question refers to a most complicated situation, I am not confident that the following answer is correct. Suppose that, under the influence of uniform stimulation in a restricted area of the retina, impulses arrive in the cortex with random temporal relations among the potentials of individual fibers. From a macroscopic point of view, it might be argued, the statistical result would be an approximately steady potential in all parts of the active region. The fact that under these circumstances action potentials in some fibers would temporally coincide with after-potentials in other fibers need not seriously affect this conclusion, since after-potentials are supposed to be weaker than action potentials. But if non-synchronized impulses establish a steady potential, then we must also expect that a steady field or current develops which spreads through and around the active area. In other words, if this interpretation were accepted, the hypothesis of pattern currents, which we have tried to verify, would be accepted at the same time, although a special assumption about the origin of such currents would be added. As has been said before, we can think of several ways in which the electromotive forces of pattern currents might originate. The problem arises only if there are such currents, and, since in this respect the outcome of the present speculation seems to agree with our own thesis, I have no reason to raise objections.

It is interesting to note that, on this basis, satiation can also be given a satisfactory explanation in terms of nerve impulses. The main point is, of course, that the term "impulses" must now be used in its wider sense, in which the fields or external currents of impulses are no less important than are the events in fibers. A current maintained by the (macroscopically) steady potential of non-synchronized nerve impulses would assume the distribution which has been discussed above.

---

* We can, of course, ask why, in experiments of this kind, no *direct* currents have been found if there are any. The answer is, first, that with most condenser-coupled amplifiers only inadequate symptoms of a steady flow can be registered; and, second, that indications of a steady flow are likely to be overlooked when there seems to be no reason why such a flow should occur. It may also not be quite correct to say that no evidence of direct currents has ever been found under such conditions. Jasper once obtained curves with short deflections upon short visual stimulation which resemble the more persistent responses in our own records (6).

It would circle around the contour at which two areas of different potential are in contact. If one of these areas is surrounded by the other, and if the surrounded region is smaller, the density of the current would be maximal in this region, irrespective of its level of activity. More particularly, it would be maximal also in a circumscribed "black" region which is surrounded by "white." Now, a steady flow polarizes the tissue through which it passes, and since the intensity of the resulting electrotonus grows with the density of the flow, the obstruction would be greatest within the area of circumscribed objects, whatever their brightness may be. This is, of course, the same explanation of satiation as has been given above.

Quite apart from questions which refer to the nature of satiation, is the present interpretation of our records actually tenable? Others are much more competent to answer this question than I am. In fact, I should like to assume a reserved attitude with regard to most explanations which have just been discussed. So far as my own views are concerned, there is a particular reason for caution. Our problem is closely related to many others. If it were really demonstrated that pattern vision is a matter of continuity physics, then we should be seriously tempted to believe that this holds also for analogous facts in other parts of psychology. Just because I realize that we are dealing with a major problem of psychophysics in general, I feel inclined to move slowly. It may be that the cortical correlate of pattern vision has now become accessible to experimentation. But we can not yet be sure that it has.

REFERENCES

1. Adrian, E. D., and Matthews, B. H. C. The Berger rhythm: Potential changes from the occipital lobes in man. *Brain*, 1934, 57, 322–351.
2. Gerard, R. W. Factors controlling brain potentials. *Cold Spring Harbor Symposia*, 1936, 4, 292–304.
3. Gerard, R. W. The interaction of neurones. *Ohio J. Sci.*, 1941, 41, 160–172.
4. Gerard, R. W., and Libet, B. The control of normal and "convulsive" brain potentials. *Amer. J. Psychiat.*, 1940, 96, 1125–1153.
5. Gibson, J. J. Adaptation, after-effect and contrast in the perception of curved lines. *J. Exper. Psychol.*, 1933, 16, 1–31.
6. Jasper, H. H. Cortical excitatory state and synchronism in the control of bioelectric autonomous rhythms. *Cold Spring Harbor Symposia*, 1936, 4, 320–339.
7. Köhler, W. *The place of value in a world of facts.* New York: Liveright Publishing Corp., 1938.
8. Köhler, W. *Dynamics in psychology.* New York: Liveright Publishing Corp., 1940.
9. Köhler W., and Dinnerstein, D. In *Miscel. Psychologica Albert Michotte.* Louvain, 1947.

10. Köhler, W., and Emery, D. Figural after-effects in the third dimension of visual space. *Amer. J. Psychol.*, 1947, **60**, 159–202.
11. Köhler, W., and Fishback, J. The destruction of the Müller-Lyer illusion in repeated trials: I. An examination of two theories. *J. Exper. Psychol.*, 1950, **40**, 267–281.
12. Köhler, W., and Held, R. The cortical correlate of pattern vision. *Science*, 1949, **110**, 414–419.
13. Köhler, W., and Wallach, H. Figural after-effects, an investigation of visual processes. *Proc. Amer. Philos. Soc.*, 1944, **88**, 269–357.
14. Lashley, K. S., and Russell, J. T. The mechanism of vision: XI. A preliminary test of innate organization. *J. Genet. Psychol.*, 1934, **45**, 136–144.
15. Libet, B., and Gerard, W. Steady potential fields and neurone activity. *J. Neurophysiol.*, 1941, **4**, 438–455.
16. Liston, M. D., Quinn, C. E., Sargeant, W. E., and Scott, G. G. A contact modulated amplifier to replace sensitive suspension galvanometers. *Rev. Sci. Instr.*, 1946, **17**, 194–198. At present, the Perkin-Elmer Corporation in Glenbrook, Conn., builds the instrument.
17. Verhoeff, F. H. A theory of binocular perspective. *Amer. J. Physiol. Optics*, 1925, **6**, 416–448.

DISCUSSION

DR. LASHLEY: I attended the dedication, three weeks ago, of a bridge at Dyea, Alaska. The road to the bridge for nine miles was blasted along a series of cliffs. It led to a magnificent steel bridge, permanent and apparently indestructible. After the dedication ceremonies I walked across the bridge and was confronted with an impenetrable forest of shrubs and underbrush, through which only a couple of trails of bears led to indeterminate places. In a way, I feel that Professor Köhler's position is somewhat that of the bridge. He has, as you are all aware, presented only a very small part of an enormous amount of material which he has accumulated and he has not done justice to the completeness and ingenuity of the field theory which he has developed. This field theory is at the present time, I am sure, the only set of concepts which ties together the phenomena of visual perception and gives meaning to them. Nevertheless, I am at a loss to see where further development of the theory will lead.

Sensory effects conveyed to the cortex are certainly in the form of discrete nervous impulses. The field theory is based upon their spatial distribution in the cortex and the electrical phenomena to which they give rise. Motor activity is also certainly the product of discrete efferent impulses to the muscles. The neurological problem is in large part, if not entirely, the translation of the afferent pattern of impulses into the efferent pattern. The field theory in its present form includes no hint of the way in which the field forces induce and control the pattern of efferent activity. It applies to perceptual experience but seems to end there. Dr. McCulloch has been inclined to regard the phenomena dealt with in field theory as smear effects to be disregarded in the development of our concepts of neurological function. Dr. Köhler, on the other hand, has minimized the importance of neuronal discharges in the integrative functions of the cortex. I hope that we can find some way of bringing these two points of view together, because I feel that both deal with genuine phenomena which are significant for understanding of behavior. I wonder if Dr. Köhler would say a word as to the road beyond the bridge?

DR. KÖHLER: The question "What next?" does not disturb me much at this moment, because if my evidence were accepted, we should immediately have to deal with almost too many problems. Some, as Professor Lashley rightly says, refer to the relation between our well-established knowledge of nerve impulses and the thesis that cortical function is, to a degree, a matter of field physics. Since my beliefs concerning nerve impulses are fairly orthodox, I do not wish to deny that further investigations will have to clarify the connection between impulses and steady potentials. In this respect, it seems, we need not expect great difficulties so far as the relation between afferent impulses and cortical currents is concerned. It may be a harder task to discover how a current pattern in the cortex gives rise to centrifugal impulses, and how the distribution of such impulses depends upon that pattern.

Always assuming that further experimentation will verify my main thesis, unemployment in this field is also not to be expected because of the following questions: Is it true that the spatial characteristics of visual patterns depend upon functional relations within the corresponding pattern currents? If it is true, what precisely are the functional relations which determine the shape, the size, and the grouping of visual facts? Since visual space has three dimensions, can a satisfactory account of visual patterns in three dimensions also be given in terms of pattern currents? What, in such terms, is a stroboscopic movement? Is tactual and kinesthetic space a matter of direct currents as visual space seems to be? Is it right to suggest, as I did many years ago, that cortical currents inscribe their own patterns on the tissue through which they pass, and thus establish memory traces?

Once the existence of pattern currents has been proved, all these questions and many others will have to be answered. Under the circumstances, I am less afraid of not having a sufficient supply of further questions than I am of not finding men who can answer them. For such men will have to know not only psychology and neurophysiology but also parts of physics with which, unfortunately, not many people are now familiar.

DR. VON NEUMANN: I am repeating a question I asked earlier, and perhaps Dr. Köhler will give his answer to it. The question is whether these figural after-effects might not be interpreted as a reorganization of the visual picture effected by the visual brain. It would not be implausible to assume that those regions of the picture recognized as relevant have an inflated representation, and those far less relevant, a contracted representation; and therefore that the detail will be greater around contours than away from them. If this is true, it necessarily leads to distortion of the field in which the region around the contour is magnified, and the other regions demagnified, and would lead to displacements in the field very much like those demonstrated by Dr. Köhler.

DR. KÖHLER: So far I have said little about the explanation of figural after-effects in terms of pattern currents and their electrotonic action. Since Dr. von Neumann now raises this issue, I will try to indicate in what direction the explanation seems to lie. First of all, extension in visual space is surely not determined by geometrical conditions alone. For instance, if the cortical counterparts of three equal visual objects are separated by two equal distances in the brain, the corresponding visual distances need not appear equal. They would appear equal only if the specific conductivity on both sides of the central cortical object were the same. In this case, the distribution of the pattern currents would be symmetrical on both sides. But any factor which disturbs this functional symmetry would make the two distances look different. Thus, if the conductivity of the tissue were lower on one side of the central object, the flow on this side would be weakened, and the flow on the other side would be intensified, as follows from Ohm's law. Visually, this asymmetry would be represented by a corresponding asymmetry of the two distances. The distance across which two cortical objects are less intensely related would appear longer, and the distance across which functional interrelation is intensified would appear shorter. Now this is exactly what happens when, before the presentation of the three objects, an I-object is shown in the area between two of them. The current of this object raises the impedance in its place and in its immediate environment. As a consequence, the currents of the objects which are afterwards presented must assume an abnormal distribution. There will be more current on the unaffected side, and less between the cortical objects which are neighbors of the affected region. Functionally, these objects are therefore less closely related than are the central object and the third object, and in the visual field the distance between the former appears greater. This means, of course, that the cortical correlates of facts in visual space are functional rather than geometrical relations. Geometrical relations in the brain determine spatial relations in vision only to the extent to which functional relations in the cortex depend upon those geometrical facts. The main point is that such interrelations do not depend on geometrical facts alone. Any change in the conductivity of parts of the visual cortex will affect visual sizes inasmuch as that change causes distortions of the currents which pass through those parts and their environment. It seems that vision does not distinguish between redistributions of currents which are brought about in this fashion and certain others which follow actual changes of retinal images and their cortical counterparts. So far as vision is concerned, only the redistributions as such seem to count.

It follows that there is no constant geometry of visual space. Visual space depends upon the visual processes which happen to occur at a given time. In a way, such processes build their own space from one moment to another. This cannot, of course, prevent us from formulating the general principles according to which the space-building is done in each case.

Let me add a further remark about the complications with which we are here confronted. One is tempted to say that prolonged presence of an I-object lowers the conductivity of its cortical area and of adjacent regions. Actually, the change appears to be mainly an increase of the polarizability of the tissue in question. From a functional point of view, the difference is quite important. For, if the conductivity of an area is lowered, the intensity of a current which passes through this area is at once decreased. On the other hand, if the same area assumes a higher degree of polarizability, a test current will be weakened only as its own flow polarizes the area to the high degree which is now possible. This takes a certain time. For this reason, the size of a figural after-effect depends not only upon the intensity of the "satiation" which has been established in a certain region but also upon the way in which test objects are shown. When the exposure of these objects is quite short, say, a small fraction of a second, their displacement is smaller than it is when they are shown for longer periods. In this sense, test objects play an active role in establishing figural after-effects. When test objects are presented for several seconds, one can sometimes actually see how the figural after-effect grows during the first few moments.

DR. LORENTE DE NÓ: There are a few points that I would like to mention. As a nerve physiologist, I am, of course, inclined to explain everything in terms of the observations one makes with nerves, and I don't encounter, in my mind, any great difficulty in explaining figural after-effects as a consequence of the activity that has gone on in the cortex during the period of exposure. For example, one thing that must play an important role in every kind of visual phenomenon is the fact that there is no point-to-point relation in a neuro-anatomical sense. It is a physiological projection, not an anatomical one, because when the afferent fibers from the geniculate body go into the cortex, they go through very long distances which are to be measured in millimeters. A fiber may respond to a rod in the retina and may innervate millimeters of length of the cortex, and there is a partially shifting overlapping of the fibers. If, therefore, we are to have a point-to-point projection, since a point of the periphery is going to correspond physiologically to a zone in the cortex, there must be something to concentrate the activity, to channel the impulses upon particular pathways. This activity may leave a change in the fibers of the nerve cells which will affect the transmission of the impulses arriving later, and this may continue for a very long time. In this way you might explain figural after-effects. I don't know exactly how, but in my own mind, and in my ignorance of these problems, I can visualize an explanation in terms of the residual effects of the activity of neurons. Several years ago, I found a very interesting after-effect in the acoustic system, not in the cortex, but in the first sensory station of the cochlear fibers in the acoustic nuclei. I took as a test object the reflex contraction of a muscle —the tensor tympani—(the whole process took place in the lower centers in the medulla), and the type of experiment was this. A tone of a given intensity was presented to the ear, and only the duration of the tone was varied. Now, if we plot the contractions of the tensor tympani against the duration of the tone, we find that up to a certain duration of the tone the contraction increases, but that a further lengthening of the tone does not produce any further difference. The tone can be interrupted, and the muscle will be about at the same level, and a succession of pulses of tone can give the same result as a continuous tone. I don't remember the figures exactly, but some acoustic impulses produce a cycle of activity, and then the acoustic nuclei receive no more impulses. This can be described as an after-effect and might be very similar to the after-effects in the cortex.

DR. KÖHLER: I did not understand quite clearly where this effect is localized.

DR. LORENTE DE NÓ: The reflexes are in the primary acoustic nuclei and the nucleus of the tympani. The process takes place in the lowest center of the acoustic system; the midbrain and other upper centers have been destroyed. But the point is the very interesting potential changes to be recorded from visual exposure. From looking at your records, I don't see any reason why they are not perfectly legitimate records, and why we are not now in the presence of a new phenomenon in physiology. The process is of much slower duration than we have used, but we have not looked for it. As to the possibility of its being discrete impulses, I think that Dr. Gerard will agree with me that that could be a very real possibility. I would be inclined to believe there was something else, but it could be. Dr. Gerard will probably mention something about the study of potential fields now, because he found them, but I would like to mention that the processes in nerve cells and in nerve fibers are not all as abrupt as we are accustomed to believe. There are also various slow processes in nerve fibers. In an isolated and excised nerve out of the body, there is absolutely no intervention of any external thing, but one can have changes in potential with a period of one oscillation every fifty seconds or sometimes even much slower. These are probably conditioned by changes in metabolic activity and that can go at an exceedingly low rate.

DR. KÖHLER: May I repeat that an interpretation of figural after-effects in terms of impulse activities meets with a certain difficulty. When a black object has been shown on a white background, satiation proves to be maximal within the area of the black object. We can hardly derive this fact from the behavior of nerve impulses as this term is commonly understood. But, as I said before, the *fields* of nerve impulses could strongly satiate the area of a dark object.

DR. LORENTE DE NÓ: As to whether electrotonic phenomena could be blamed for the establishment of long lasting potential differences, I'd like to refer the question to Dr. Gerard to see whether he agrees that this is a possibility. Changes in the metabolism, due to intense activity, may produce a change in the potential field, and might very well be the mechanism of explanation for the potentials you are recording. In saying that, however, I do not deny the possibility of their being due to envelopes of impulses, but I doubt whether they are.

DR. GERARD: Again I see a certain amount of history repeating itself. It is somewhat to the shame of physiologists that the spontaneous rhythm of the human brain was discovered by a psychiatrist—the Berger rhythm. When these slow, ten-a-second waves were first proved to be there (and they were at first universally doubted by physiologists, until Adrian repeated Berger's work), we were faced with the problem of how potential waves lasting tenths of a second could be built of action potential spikes lasting a thousandth of a second. The first inclination of most workers was to interpret these slow waves as nothing but the envelopes of action potential spikes appropriately distributed in a regular time pattern. The argument flourished for some years, but that view has now been pretty universally given up; these are not envelopes of action potential spikes. There is all sorts of evidence against this, both in the mammal and in the isolated brain of the frog. One can get regularly repeated, distorted waves that couldn't possibly be an accidental spike pattern but would require complex reverberating impulse patterns. Yet they persist after mutilation. Any such circuits, like the rotating commutator turning on and off flashing lights, would have been destroyed in the course of the preparation, and yet the waves remain.

The ordinary slow brain waves have not really been satisfactorily interpreted, though they could well be oscillations of the somatic potential. I, personally, cannot see them as associated with discharges, per se; but if they are, then they must still represent waves of potential change primarily within a cell body, although spreading electrotonically, as Dr. Lorente de Nó has said. In that case, of course, they remain analogical, not digital, phenomena, in the sense of the discussion of the other day.

Now, again, it is not a physiologist, but a psychologist who has had the courage to try a reasonable gamble and look for his still slower changes directly in the human brain. I am much inclined to think that he has found them. The first question, then, is whether those slow potential drifts are themselves envelopes, either of action potential spikes or of altered somatic potential oscillations in groups of cells activated through impulses. Dr. Köhler was properly cautious of drawing a positive conclusion in the present state of the evidence. I would be similarly cautious, but am inclined to agree with his predilection for regarding these as something different from an envelope.

There are several experiments that he has in mind, I am sure, which will fairly rapidly resolve the problem. One quite simple one, which you may already have done, Dr. Köhler, would give some real confidence in the interpretation. That is to have both electrodes on the occipital region and see whether, on moving the light across the visual field and therefore, presumably, the steady pattern across the occipital cortex, you can get a diphasic effect, as when a potential moves along a nerve fiber.

A possible source of error occurs to me, though I think it unlikely that one can get potential changes in neural systems just from changes in vascular caliber. We were once badly fooled by rhythms from an electrode in the cat's brain which turned out to be pulsing vessels, yet not a direct mechanical pick up. There is vasodilation in the visual cortex with light, and this possibility should be kept in mind.

The fact that fairly steady and spatially extended semi-uniform potential fields exist in the nervous system normally is now, I think, established beyond equivocation; both in isolation and in vivo, both in the frog and in the mammal. That these potential fields can be altered by the physiological state of the system is also, I think, certain. The question is, as Dr. Lashley implied with his bridge story, whether one can go on from the changes in potential states to clearly attendant changes of function, whether at the level of behavior or of central awareness. I can merely give you some evidence supporting such a relation.

At a psychological level, of course, you all know that conscious effects are easily produced by passing constant currents through appropriate parts of the head. In the cat or frog brain, with a properly oriented potential one can at will initiate or stop the extremely regular rhythmic potentials, whether these are static or traveling along the hemisphere. By polarizing with currents, certainly of the order of those which could be produced physiologically, one can initiate wave trains, or can block out trains, or can change their amplitude and frequency. Recent work by Libet and myself has carried these experiments further.

Leao found that local irritation of the rabbit cortex will lead to a slowly spreading wave of suppression of the spontaneous electrical rhythm with, superimposed upon the flat base line, rather large negative spikes. One can follow the wave across the cortex with a series of electrodes and observe both the d-c and the a-c potential of a given cortical area. The functional state and the d-c potential are sharply correlated. The latter changes first and reaches greatest negativity when the brain waves die out. It then moves sharply positive, during the period of spiking, to an acute peak and more slowly subsides. Application of strychnine also leads to great d-c changes.

These potentials, which are steady or change slowly over minutes, are tremendous things. The ordinary brain wave potentials are under 100 microvolts, even convulsive ones rarely reach a millivolt; but the d-c potentials in these cases run to 30 or even 50 millivolts—a twentieth of a volt! These approach the full magnitude of the membrane potential of nerve or muscle fibers, and they show early the temporal and spatially quasi-constant variety which changes in time and characteristic positions in space. It is hard to believe they are not highly important in neural function.

DR. KÖHLER: The experiment suggested by Dr. Gerard has not yet been done, although it is on our program. Surely, when both electrodes are placed in positions which correspond to parts of the visual cortex, a moving object ought to cause a diphasic response as it passes first one and then the other electrode. We postponed this experiment because, with such a short distance between the electrodes, differences of potential would probably be quite small. We preferred to begin with situations in which our chances of obtaining reliable responses would be greater. But Dr. Gerard is quite right; if under those conditions diphasic responses were actually found, they would strongly support our main thesis.* In the same connection, it will be interesting to see what happens when brightness relations in the field are reversed. One might expect that in this case the polarity of the responses would also be reversed.

DR. GERARD: One thing worries me about that inverse experiment. If it does invert, you are pretty safe; but it might not invert, because it is the edge between light and dark which is critical, so you might get the same effect with either pattern.

DR. KÖHLER: This is indeed possible. Other experts have also told me that reversal of brightness relations may not greatly affect the responses, and that, therefore, we should not rely too much on this test. Even so, the test must be made. The result may tell us something about the origin of our responses.† In this respect, I should like to add that the electromotive forces in question need not be caused by electrotonic action, although, as Dr. Lorente de Nó has pointed out, they may be so caused. Once the currents are established, they must, of course, have electrotonic effects which are equivalent to changes of local impedance.

DR. LORENTE DE NÓ: That is perfectly correct.

DR. KÖHLER: I think that it would take too much time to explain how I derived the origin of these currents. In some way, the flow must, of course, be established by the arrival of nerve impulses in the cortex.

DR. MC CULLOCH: I have for years repeated, as I had a chance, one after another of the crucial experiments that Dr. Köhler first proposed. They are startling. They were, to me, also surprising when I repeated them fairly recently. I am puzzled by one thing that I ran into this summer, and I would like to toss it in. I would like to ask to what extent do these alterations, produced in one sense modality, affect those of another sense modality? Let me tell you a simple thing that happened. I had just pulled on one 16-inch tramping boot. An emergency arose, I kicked my other foot into a moccasin and ran. I went about with a heavy boot on one side and a moccasin on the other. When we got through, I put on the other boot and went to work, and I was disturbed for several minutes when picking up two objects, two hammers, two dowel sticks in my two hands by finding that the weights, as judged in the hands, were thrown out as you would have expected had I carried a heavy weight in that hand. Now, that is a transfer to quite a distant part of the body. I am wondering whether a lot of these effects may not be something comparable to optokinetic nystagmus. Something has appeared in our world and some mechanism has got going somewhere to offset the fixed distortion that we have in that world, like the rhythm that a sailor carries in his legs when he goes ashore. Those things can have tremendous persistence. They may be then due to what I will call a predictive circuit which has been set going due to a constancy in our world and is trying to get it regulated out. I don't know whether this experiment has been tried, but can one by putting one foot in hot water and the other in cold water effect adjustment of the hands? I don't know how far these effects go.

---

* More recently, diphasic responses have actually been recorded under such circumstances (12, page 418).

† Since this was written, a more thorough examination of the situation has made it quite clear that, so long as the "inactive" electrode is placed at a considerable distance from the visual cortex, a reversal of brightness relations in the field cannot alter the polarity of the registered currents. The results of experimental tests have confirmed this conclusion.

The other thing I would like to say will take just a minute. In this work on studying the effects of stimulating the cortex locally you observe, with a direct-coupled amplifier, the area you have just stimulated, and you find that it is electrically negative to other regions of the cortex and that its threshold, as judged by the local response to a small signal thrown into it then, is lowered. If you take strychnine and put it on an area of a deeply narcotized animal's cerebral cortex, there are no little spikes that you can detect with the maximal gain of the amplifiers and no signals kicking around that area just strychninized. Then the area begins to climb electrically, many millivolts negative, and, as it goes up, the cells in it begin to discharge. At its height of negativity come the fully developed strychnine spikes. I don't think there is any question but that one gets these effects every time when one excites the brain, whether by afferent impulses, or by direct electrical or chemical stimulation. If you drive an area hard, it becomes and remains electrically negative during the period of facilitation. I think this fits in with Dr. Köhler's findings.

DR. KÖHLER: It is a most interesting question to what degree after-effects occur in parts remote from the center of original satiation. No transfer from one sense modality to another has so far been observed. Our very technique in testing kinesthetic after-effects seems to prove that there is little or no transfer from one hand to the other. This does not, of course, prove that more remote parts of the brain are never influenced in this fashion. After all, if visual currents can reach the surface of the head, they are likely to spread quite far within the brain. The same would probably be true of currents which originate in the postcentral area. It is therefore possible that remote after-effects will be discovered in the future.

Dr. McCulloch's observation refers to transfer within a given modality. After carrying different weights on his feet he found that objectively equal weights appeared different to his hands. It seems to me that, as a matter of principle, this effect allows of two different explanations. Either this actually is a case of transfer; or the effect is caused by the "balancing reflexes" which, under such conditions, affect even remote muscles. If for such reasons the two arms and hands receive different innervations, new localized after-effects may, of course, soon be established in these parts.

DR. WEISS: What would happen if in your experiment the line were yellow, let us say, on a blue background and they were both of the same luminous intensity? Would you expect the same electric phenomena?

DR. KÖHLER: So far as pattern currents are concerned, no such experiments have been made. Figural after-effects, on the other hand, have been obtained when the object and its environment had the same brightness, and differed only as to color. I am under the impression that such effects tend to be weaker than those obtained with striking differences as to brightness.

DR. LIDDELL: I would like to ask Dr. Köhler a methodological question of some interest to me which also relates to Dr. Lashley's preoccupation. Here again I see, in my own case, history repeating itself, because in Pavlov's ill-starred attempt to explain neurophysiologically (the neurophysiology being made up in his head) traces from the irradiation of negative tactile conditioned reflexes to adjacent spots of the skin, he claimed to have found evidences of irradiation in the acoustic analyzer also. We have confirmed his results on irradiation in the tactile analyzer. In his physiological speculation, he spoke of internal inhibition and its irradiation. We remember that the neurophysiologist of Pavlov's time was concerned with the inhibition of the heart by the vagus nerve, and Sherrington's reciprocal inhibition, and now there are the various manifestations of cortical suppression of activity. I would like to ask Dr. Köhler how he proposes to proceed, in going from sense modality to sense modality, having selected a particular neurophysiological manifestation, namely, these constant currents? I think those of us dealing with such detailed phenomena in conditioned reflex behavior, which are practically identical with Gestalt phenomenology, agree that his approach will be helpful to us working in our field of conditioned behavior. How do you propose to follow this clue of the slowly fluctuating cortical potentials when you change over to the kinesthetic and tactile fields?

DR. KÖHLER: I do not yet know how our interpretation of pattern vision is to be applied to other sense modalities. To be sure, the task may be easy in the case of passive touch, where the sensory surface seems to be projected upon the cortex more or less as it is in vision. Under such circumstances, the spread of pattern currents depends on spatial configurations in the cortex which are, in each case, related to the distribution of stimulation on the sensory surfaces. In kinesthesis, however, this is not generally true. Of course, while contraction of the muscles of the left hand causes processes in one part of the cortex, contraction in the right hand has the same effect in another part. But when subjects perceive the width of an object which they hold between the thumb and the other fingers, it seems to be the degree of muscular tensions on which their impression of the width depends. For if the width of the object is changed, the same muscles are still at work, and only the tension in these muscles varies. Obviously, it is a major theoretical task to discover how a variation in the intensity of certain cortical effects can act as though a cortical distance had been varied. I have not been able to concentrate on this problem because the investigation of visual currents takes so much time.

DR. LINDSLEY: I can only marvel at Professor Köhler's many beautiful demonstrations of figural after-effects. I am wondering whether he has had an opportunity to study patients with scotomatous areas in which there is a fairly fixed projection of the defect and to take note of how that might modify the possible figural after-effect.

Something that has bothered me, I suppose largely because of thinking about electroencephalography, is, how the effects, which are presumably manifest in the striate area, are picked up to this extent on the head over what must be predominantly areas 18 or 19. How small potential changes are transmitted in this fashion is a little difficult to conceive. Also I still have a feeling that these potential changes might just possibly be galvanic skin phenomena, or polarity potentials from the eyeballs. The polarity potential of the eyeball may be of the order of millivolts, and we know that in brain wave recording, its effect is often detected at least as far back as the fissure of Rolando. I can't quite see how this could be picked up on the back of the head, but it is very easy to be certain. One has only to put electrodes around the eye to pick up eye movements in any direction. I think it would be very desirable to have records of the polarity potentials of the eye and at the same time from the surface of the scalp to check the possibility of galvanic skin phenomena. With such tests, one could rule out other possible peripheral effects as well.

DR. KÖHLER: There have been no experiments with brain-wounded subjects. Swarthmore has no medical school. But certain observations made by others seem to relate figural after-effects to pathological phenomena. If I understand correctly, Bender and Teuber find that, in the visual field of patients with circumscribed (cortical) scotomas, test objects shown in the neighborhood of the injured regions are wrongly localized; they are displaced away from these regions. In this respect the functional characteristics of injured areas seem to resemble those of strongly satiated areas. One feels tempted to assume that, in both cases, the impedance of the crucial regions is increased.

Dr. Lindsley's question whether figural after-effects in vision could partly be caused by peripheral factors is not quite easy to answer. To a minor degree, this may be the case. But, generally speaking, the after-effects are far too great to be explained by changes in the width of the pupil or by small eye movements. After satiation with certain I-patterns, subjects may find a line with the objective length 7 centimeters equal to an unaffected line with the length 5.5 centimeters. This may happen when both lines lie near the fixation mark, and when the subject observes from a distance of about 2.5 meters.

I quite agree with Dr. Lindsley's suggestion that, in the future, the position of the electrodes be widely varied.

DR. KLÜVER: In connection with Dr. McCulloch's question concerning possible relations between different sense modalities you said, if I understood correctly, that there is no transfer from kinesthetic to visual effects. Are there transfer effects in the opposite direction?

DR. KÖHLER: To my knowledge, no transfer of figural after-effects from one sense modality to another has so far been demonstrated.

DR. KLÜVER: If I remember correctly, there are reports in the literature indicating that certain forms of visual stimulation produce postural changes. I do not recall whether the subjects used in these experiments were normal individuals or patients with labyrinthine disturbances. The subject was required to stand before a wall covered with alternating black and white stripes. After prolonged observation of a wall with stripes deviating from the vertical the subject's own body deviated from the vertical. I am wondering whether effects of this kind are related to the after-effects you have described. I am also wondering—and this may seem far afield—whether you consider it possible that the phenomena so frequently observed by Goethe with closed eyes involved figural after-effects. He often saw leaves or flowers undergoing changes in size and shape. In commenting on these phenomena he himself called attention to his previous long-continued observation of such materials during his study of the "metamorphosis of plants."

DR. KÖHLER: If I am not mistaken, Dr. Klüver refers to what we call "normalization." In a room in which all normally vertical and horizontal contours are turned by the same angle, an actual vertical line appears tilted. This phenomenon, which has recently been investigated by Asch and Witkin, differs from analogous figural after-effects by its often enormous size and by its temporal characteristics. One does suspect that the two facts are somehow related; but I have not yet been able to discover their actual connection.

Brain and Intelligence

WARD C. HALSTEAD
Departments of Psychology and of Medicine,
University of Chicago

It is apparent to members of this symposium that the task of relating behavior and brain functions is indeed a formidable one. I should prefer to think that this is especially true for the class of behaviors known as intellect in man. But it seems that it is also true for the simplest unit of ordered behavior recognized by psychologists. As long ago as 1912, Yerkes (36) reported that he was able to train the lowly earthworm to form a direction habit in exiting from one arm of a T-maze. The left arm of the maze contained at its entrance a strip of sandpaper which signaled the presence of an electric grill, located just beyond. The other arm of the maze opened directly on a dark, moist environment. Yerkes started his worms at the base of the T several inches from the choice point. He found that the habit of turning toward the right arm of the T-maze (thus avoiding the grill) appeared in twenty to one hundred trials. A three-week rest period yielded no loss of the habit. Surgical removal of the cephalic ganglions or "brain" of the earthworm did not destroy the habit. In fact, the anencephalic worms performed quite well until the anterior segments began to regenerate. With the coming of a new "brain" the habit degenerated. Where were the traces of the habit during the interval between the first and second brain?

A satisfactory answer to one form or another of this question has commanded the interest of a considerable segment of biological science during the last half century. Some of us are not impressed by the progress that has so far been made in our respective disciplines towards finding an answer which will account for the behavior of Yerkes' earthworms. I can only ask your indulgence as I discuss somewhat analogous problems as they arise at the level of man.

It is virtually impossible to date accurately the beginnings of our topic. According to the late Professor Henry Breasted (4), the term "brain" is recorded for the first time in human speech in the Edwin Smith Surgical Papyrus, which was transcribed about 1700 B.C.

from a text which may go back to 3000 B.C. This early usage of "brain" may also be close to the beginnings of written communication of intelligence. As Breasted relates of the commentary incorporated gratuitously into the Treatise by an unknown Scribe:

> This commentary is to us a priceless revelation of the meaning of many words and terms which would otherwise have remained hopelessly unintelligible, coming down to us, as they have done, from an age when surgery was first creating its technical terms some five thousand years ago. The study of the terms is a fascinating revelation of the human mind struggling with the first stages of science-building, when even the terms it needed did not yet exist, but had to be created, and we watch the process of their creation actually going on. . . .
>
> Like the modern scientist he (the ancient) clarifies his terms by comparison of the things they designate with more familiar objects: the convolutions of the brain he likens to the corrugations on metallic slag. . . . A puncture of the cranium is like a hole broken in the side of a pottery jar . . . (page xv).

Not only does the Smith Papyrus contain the earliest known anatomical, physiological, and pathological descriptions; it also provides evidence of some systematic observation. Quoting Professor Breasted again:

> The observation of effects on the lower limbs of injuries to the skull and brain, noted by the ancient surgeon with constant reference to *that side of the head which has been injured,* shows an astonishingly early discernment of localization of function in the brain—and observation which has been more fully developed by modern surgeons only within the present generation (page xv).

There is no record as to how the early Egyptians acquired their limited insights, however. The experimental method was probably yet to be born. We know only that, regardless of ancient interests, systematic studies attempting to relate brain and higher mental processes were first recorded in modern times.

A French psychologist, Alfred Binet (1857–1911), was the first to succeed in some measure in bringing an operational or quantitative approach to the problem of intelligence. His contributions are probably inadequately reflected by the well-known I.Q. test which bears his name. His versatility, keen insight, and intellectual brilliance were of such magnitude that he could study their opposites in man with penetration. Goddard (3) wrote of him in 1916: "After ten years of the Vineland laboratory, it is surprising to find how little we have found that Binet had not already discovered."

Binet (3) distinguished between intellectual "activity" and intellectual "level." He wrote: "Who has not encountered persons who busy themselves with a host of questions, have a great deal of information, speak of everything with warmth and an inexhaustible supply of words, are fertile in views, hypotheses, distinctions, neologisms? Very often they deceive as to their true value. They are thought very intelligent, while in reality they possess only intellectual activity."

Farther on Binet emphasized intelligence as adaptation: "One must remember that the faculty of adapting oneself is the property of the intelligence and that the power of adaptation is the measure of it; it is evident that from this point of view any confusion between the activity and the level is impossible."

Binet viewed intelligence through the eyes of a biologist. He recognized both genetic and environmental factors as contributors, but there is little question that he assigned the intellectual level to the former.

As to the scale which Binet produced for measuring intelligence, and its subsequent modification by Terman, I suspect that it is a better measure of intellectual activity than of intellectual level. This, of course, is not the outcome that Binet had hoped for. But, as Hebb (14) has convincingly demonstrated, the I.Q. may or may not be changed and may be maximal for the scale with the major part of both frontal lobes (about 30 per cent of the cerebrum) absent.

Binet's reaction to this recent finding would, I am sure, have been to make better tests. His clinical insight was too keen to permit the easy inference—which unfortunately is being made rather commonly today—that the frontal brain has nothing to do with intelligence.

Binet's distinction between intellectual "level" and intellectual "activity" has largely been overlooked or forgotten by contemporary students of the problem. There is a growing tendency among some psychologists to describe by factor analyses the factorial composition of scales invented to reflect a wide range of intellectual activities. The obtained factors are then equated with intelligence. It is apparent that the resulting definition of intelligence will depend to a considerable extent upon what activities are scaled and the particular form of factor analysis employed. As a result of these and other influences, there is no generally accepted meaning of the term intelligence. Like the early Egyptians, we are still building our basic scientific terms.

Since known biological correlates of intellect are lacking, it has not been possible to make satisfactory estimates of intellectual level independently of performances or activities. That this lack leaves the problem of intelligence and certain other behavioral problems "suspended in mid-air" is apparent when we recall that Binet did not hesitate to discuss the "great intellectual activity" of some imbeciles (3). I would like to state parenthetically that, if all other reasons were lacking, which they are not, the continuing need for parameters other than performance with which to specify level of behavioral organization is sufficient to link the future destinies of psychology and basic biology. Lest some of my fellow psychologists charge me with being a "reductionist," I hasten to point out that the gain sought is in classes of information. Two independent classes are better than one.

STRUCTURAL CORRELATES OF INTELLECT

From a morphological point of view, the evidence available indicates that the human brain at birth is characterized more by its communalities or similarities to other human brains at this age than by its differences or uniquenesses. Nature seems to have adopted a single basic architectural plan for the human brain which is delivered at term in a remarkably constant state of incompleteness. Many of the details of this incompleteness were accurately described prior to 1900. For example, Flechsig (8) found that different areas of the cerebral cortex acquired their myelin sheaths at various periods of development, in a definite order. Flechsig's map of this process is similar to that of Vogt shown in Figure 1. Note that at birth the myelination process is relatively well advanced in the primary sensory projection areas for vision, touch, and hearing and in the primary motor cortex, as shown by the heavy stippling. Note further that the degree of myelination shades off gradually (lighter stippling) into surrounding areas until it is conspicuously absent in considerable portions of the parietal, occipital, and frontal lobes. Substantial confirmation of these essential features of Flechsig's map has been reported in recent years by Jakob (17) in Argentina, by Conel (7) in the United States, and by others.

Conel has reported the results of detailed study of several parameters of development in addition to myelination, namely, (1) the width of the entire cortex and of each horizontal layer, (2) number of nerve cells, (3) size of nerve cells, (4) condition of the chromophil substance, (5) neurofibrils, (6) size, compactness of structure, and length of the processes of nerve cells, (7) pedunculated bulbs, and (8) size and quantity of exogenous fibers. It is of interest that all of these criteria appear to be correlated and that any one, such as myelination, serves as a reasonably good index to that stage of growth of the brain.

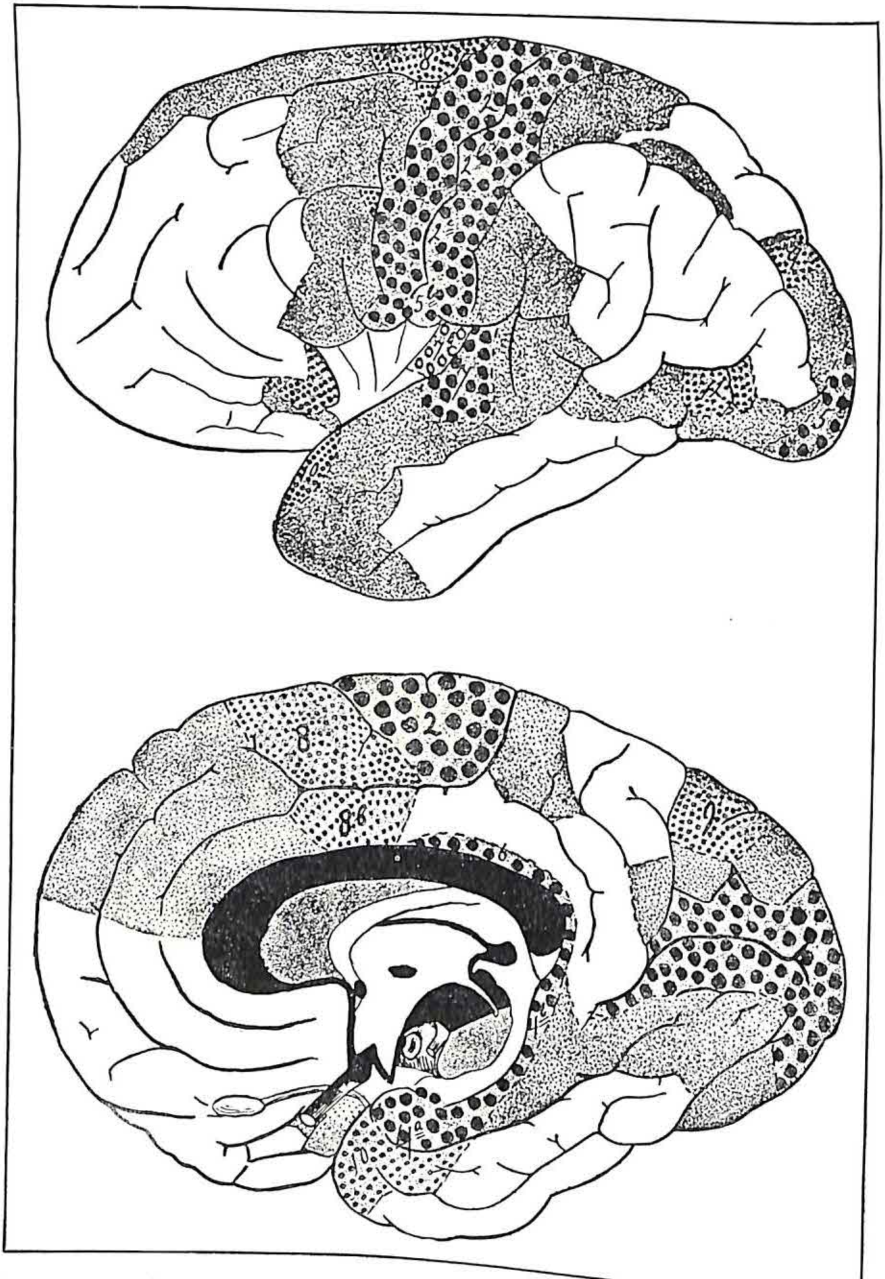


FIGURE 1. The myelination of the human cerebral cortex at birth. The fields which myelinate first are indicated by coarse dots, those next to myelinate by fine dots, the next by a gray color, while the last field to myelinate are left white. O. Vogt. (After Kappers, Huber, and Crosby, 18.)

An important critical attack upon the behavioral significance of differential rates of myelinization in the brain appeared from the University of Chicago Press in 1903. It has received but little attention, possibly because of a rather non-specific title, *Animal Education* (33). Its author was at the time an assistant in experimental psychology in the Department of Psychology of the University. His name, now well known for his behavioristic psychology, is John B. Watson. Watson employed careful training methods to ascertain that the white rat at the age of 24 days can learn associations achieved by the adult rat. When he made histological comparisons of their nervous systems, however, he found only 20 per cent of the neurons myelinated in the 24-day rat. He concluded that the degree of myelinization was not correlated with learning ability in the rat.

The cortex is usually analyzed into six layers, the dominant elements of which are shown schematically as though opened up in space in Figure 2. Note that the layers are numbered at the extreme left of the drawing. The layers are commonly designated further, from without inward as follows: (1) the molecular layer containing but few neurons, (2) the outer granular layer, (3) the external layer of pyramidal cells, (4) the inner granular layer, (5) the internal layer of pyramidal cells, and (6) the layer of fusiform cells. This arrangement is the basic plan of most of the cortex in man, comprising the isocortex both at birth and in the adult. Thus far not a single function can be attributed with certainty to any of these layers. However, Bailey (1) directs our attention to the fact that wherever there is a primary projection of sensory impulses to the cortex the granular layers appear to predominate. It is well known that the precentral motor cortex is characterized in the adult by the absence of the granular layers. Whether this suggested correlation has any value for analysis of function remains to be seen.

There is one aspect of the general problem where ontogenetic considerations might have been of considerable aid. This is in connection with nomenclature of the brain. Our misleading term "prefrontal lobe" would not have occurred had the frontal lobe been designated ab initio in terms of those anterior structures which differentiate last, for example, through myelination.

In addition to the evidence from developmental anatomy of the presence of differentiated areas in the brain, supporting evidence had been obtained during the nineteenth century from (1) gross dissection of the brain, (2) microscopic studies of brain sections which stained differentially, (3) electrical stimulation of the surface of the brain in lower animals and in man (conscious individuals), (4) experimental lesions in lower animals, and (5) clinical observations of the effects of injuries to various parts of the brain in man. However, none of these lines of evidence had cast light upon the functions of the unstippled areas in the slide which we have just viewed. Hence, these late myelinating areas came to be known as "silent" areas. It was suspected by many that therein resided the soul. For some, this included the mind, for soul and mind were used interchangeably even in some textbooks of physiology as recently as 1900.

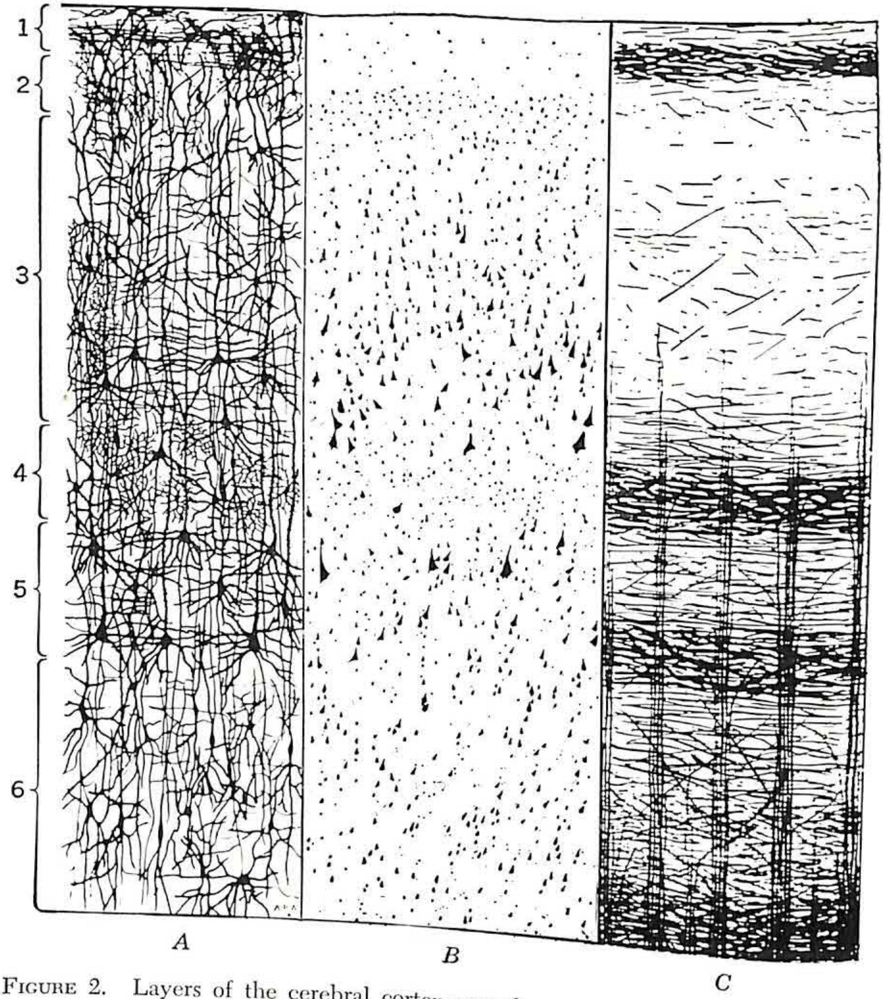


FIGURE 2. Layers of the cerebral cortex according to elements revealed by differential staining. (After P. Bailey and G. von Bonin. *The Isocortex of Man.* Urbana, Ill. University of Illinois Press, 1951. By permission of the authors.)

LESIONS IN BRAIN STRUCTURE AND ALTERATIONS IN INTELLECT

While we have thus far seen no close relationships between the cellular components of the brain and discrete units of behavior, there remains the possibility that larger masses of brain elements may be directly correlated with larger or "molar" units of behavior. Whether we select man or a lower animal as our research subject, there are several considerations which bear upon the outcome. Some of the more important ones are indicated below.

Specification of a Unit of Behavior. It is apparent from our discussion of Binet's attempts to specify units of intelligence that the task is very difficult. There simply is no general agreement as to what is meant by intelligence. On the other hand, there is widespread professional and popular belief that intelligence is one of man's highest attributes. Darwin made intelligence one of the important parameters in natural selection which account for man's strategic position in the animal kingdom. He who would explore the relations of intelligence to brain structures is first faced with the task of setting up an operational definition of intelligence. Lashley resolved this part of the problem for the rodent by using habits of various degrees of complexity as his units of intelligence. The latter are disturbed by cortical lesions according to his well-known principle of mass action. But whether the principle covers alterations in "level" of intelligence as well as in "activities" is not yet clear.

Köhler adopted somewhat more permissive units of behavior for his apes. But thus far he has avoided bringing his conceptual units of behavior to direct test against brain lesions.

If it be assumed (and this writer is unwilling to make the assumption) that the I.Q. yielded by the Stanford-Binet intelligence test is an adequate test for intelligence in man, then a maximal adult I.Q. for the scale is possible with about one-third of the total cerebrum lacking. It is possible that the principle of mass action holds for the I.Q. and that the threshold lesion is somewhere in excess of 30 per cent of the total cerebrum. However, there is good clinical evidence, and some experimental evidence to be considered later, that intelligence level in the sense of adaptive capacity is lowered by lesions in the brain of considerably less than 30 per cent.

Specification of the Lesion. It is probably safe to say that no brain lesion has ever been completely specified. To do so would require knowledge that simply is not yet available. Mapping of the lesion by histological techniques, usually possible only with lower animals, maps only a visible, structural feature of the lesion. Details of ultra-structure, metabolic aspects of synthesis of nucleo-proteins, altered circulatory dynamics due to such considerations as changes in vascularity or in sludging of the blood, specific chemical depletions or alterations associated with injury or removal of brain tissue, the temporal course of the lesion, and the extent of the personality trauma are all relevant but usually unknown factors. Until these matters can be taken into account, we cannot be certain that we are juxtaposing appropriate units of behavior and structure. It has been suggested that the presence of residual pathological tissue in some neurosurgical patients will account for their lowered level of intellectual functioning. This, I am afraid, represents a special kind of mysticism since these same patients are frequently asymptomatic in terms of mental functions until a surgically elaborated lesion for removal of a brain tumor is induced. The further argument that such pathological tissue produces bio-electrical disturbances in the form of asynchronous firing of cellular elements is not convincing in view of many well-known cases of such asynchronies with intact mentation who have some form of epilepsy.

Degree of Linearity of Our Behavioral Scales. Do our psychological scales measure equally well throughout the total range of a given capacity? This is a question which has plagued designers of mental tests since the days of Binet. There is much evidence in the literature of psychology which suggests that, for the normal population, our mental scales are more successful and more linear in scaling the intermediate range of ability than the extremes. There is certainly reason to think that this holds in the realm of patient populations; in fact, the non-linearity at the extremes of the distribution may be even more marked here. I believe that Hebb (15) failed to take this factor adequately into account when he evolved the principle that: "The case in which the fewest symptoms follow a known surgical removal is likely to be the one in which the truest picture of the effect, or lack of effect, of the removal is given." One investigator has interpreted this "principle of economy" thus: "If you do a series of patients and eleven patients show something and the twelfth one does not, the tendency is always to say that the twelfth one was somehow different and we neglect the fact that he was really the important one and the other eleven were something else again and did not enter into the situation."

I must take exception to this reasoning. We can well imagine the sad state of pharmacology, for example, if this principle were generally invoked for assessing the toxicity of specific drugs. The important question at issue is whether our methods, psychological, clinical, or otherwise, describe each of the twelve individuals with the same fidelity. We can beg the question, and it frequently is begged, either for or against the exceptional case. But when we do beg the question, we lose whatever protection against error our scales afforded and revert to a mere chance basis for being right or wrong. The practical answer, from my point of view, is to restrict the application of our scales to ranges of ability where they can be shown to be reasonably linear and hence capable of reflecting gains or losses, due to some focal intervention, in quantitative terms. As scientists we cannot move faster than our instrumentation permits.

Learning Effect in Repeated Measurements. Another factor which tends to limit the sensitivity of mental tests in studies being carried out in increasing numbers today is the factor of learning. This factor can operate as an unknown even in apparently elegant research designs. Where a battery of mental tests is applied before and after some focal procedure, the factor of learning may mask a true deficit or accent spuriously an apparent benefit. The effect of learning is extremely difficult to control or estimate since our only means at present involves repetition of the tests in question and this in turn induces a further increment of learning of unknown magnitude. It is probably not adequately controlled by using an unoperated control group which takes the battery of tests twice or more. Here, learning effect may be compensated for more or less by monotony or boredom—even in psychotic individuals. The operated or experimental group, on the other hand, may bring increased motivation of a subtle type to bear in the post-operative testing. In other words, the character of the task (psychological demands) may have changed as a function of the operation without revealing the magnitude of a learning effect.

The degradation process in learning offers a further methodological complication. The associated traces may not remain fixed in locus. As long ago as 1929 Lashley (20) pointed out that simpler problems offer difficulties which are not much greater for animals with brain lesions than for normal ones; and, correspondingly, the difficulty does not greatly increase with increasing magnitude of brain injury. One of the most conspicuous features of learning is the reduction of initially complex behavior to the level of relatively simple habits. If much of this reduction occurs during the pre-operative test, then *the persisting habit is simpler than the apparent complexity of the test battery.* As in the case of the Yerkes earthworm (36), the habit may shift in its dependency upon specific neural structures during its temporal course. There is as yet no satisfactory solution to this methodological problem. Perhaps the time will come when we can specify the factor of learning in terms of its chemical and physical concomitants, or else employ equivalent test batteries that measure the same functions without transfer effects. In the meantime, our experimental designs should provide for one experimental group which is tested for the first time *after* the focal procedure in addition to an experimental group tested before and after, and appropriate control groups.

BIOLOGICAL INTELLIGENCE

Man's strategic position in the animal kingdom derives in part at least from the unusual range and quality of the perceptions of which he is capable, from his capacity for controlled adaptability, and from the degrees of freedom through which orientation can be achieved and maintained. Psychologists will see in this statement some of the classical chapter headings in psychology: perception, emotional behavior, learning, thinking, and intelligence. Possibly less obvious is the extent to which these constructs comprise common elements, partially overlapping elements, and unique elements. Yet the psychologists' view of man as organism will attain the scientific ideal of parsimony, in terms of operational descriptions of necessary and sufficient conditions, only when such details can be accurately specified. Psychologists may take pride in the fact that some of their colleagues have made substantial progress in developing mathematical tools to aid in this abstracting process. Factor analysis appears to be such a tool (see Spearman, 27, 28, 29; Thurstone, 31, 32). While its proponents have for the most part limited themselves to applications within highly restricted domains, such as the "psychological," there is good reason to believe that the method of factor analysis is not inherently so limited. Thus there is basis for hope that suitable metrics may be found whereby more inclusive biological domains of the organisms may be ordered by this method.

Recent progress in the analysis of human intelligence appears to give substance to this hope (11, 12, 19, 29). Laboratory studies, supported by factor analyses of the data, of normal individuals and patients with brain lesions have suggested a four-factor theory of human intelligence which involves several domains of the organism. The four factors appear to be differentially represented in the cerebral cortex, and hence are interpreted as being biologically important to the survival of the organism. This biological intelligence seems to bear directly upon the capacity of man for controlled adaptability, while, over a considerable range, psychometric intelligence, as measured by the intelligence quotient, does not (11, 12, 14). It is possible that suitable methods can be devised for measuring biological intelligence in infrahuman animals, although the necessary investigations largely remain to be carried out.

Three of the four factors isolated in analysis of biological intelligence appear to have direct neural correlates. The *A* factor, or capacity for abstracting universals or rational concepts, seems to be a general property of the cerebral cortex in man that is maximized in the cortex of the prefrontal lobes. The cerebral power factor *P* also seems to be represented throughout the cortex, but again is maximized in the cortex of the prefrontal lobes. These two factors are disturbed together in unilateral or bilateral lobectomies of the frontal lobes wherein the cortex is removed along with the white matter. Both factors are spared in lobotomies, however, wherein the white matter is destroyed, leaving the frontal cortex essentially intact. On the other hand, spontaneous mild atrophy of the frontal cortex in middle age may first impair the *A* factor and only later as the atrophy progresses come to impair the *P* factor. Such individuals represent the obverse of clinical depressions. There may be no flagging of intellectual drive and ambition in the face of mounting judgmental incapacity. The mild clinical depression, probably as a direct function of the substrate, is associated with an acute drop in the *P* factor with only a more gradual restriction of the degrees of freedom for the *A* factor. It seems quite probable that analysis of neural nets will cast important light upon the special neurophysiology of the *A* and *P* factors (1, 23, 35). Theoretical advances in model building have already been forthcoming. It might be mentioned that the recently announced science of cybernetics directs much of its attention to elucidation of the *A* factor (34). It is of interest that designers of digital and analogous computing systems as models of the nervous system apparently assume a constant or critically regulated power supply for their models. Since it is quite unlikely that man is a closed energy system, cybernetics will undoubtedly have to make provision for the *P* factor as a dynamic variable.

The *D* factor is the particular modality or avenue through which intelligence is situationally exteriorized. Depending upon its degree of organization, it is formative upon the behavioral outcome. Influences organized around either perception, response, or both may affect or even control the variance. A verbal agnosia resulting in alexia may deform biological intelligence on the receptive side for written symbols while a verbal apraxia resulting in agraphia may impair the expression of biological intelligence via written symbols. Some of the agnosias and apraxias seem to result from focal lesions in the cerebral cortex (Nielsen, 24). The cortical representations of others are as yet unknown. Any truly operational description of intelligence must include among its necessary and sufficient conditions provision for an organized avenue or modality for exteriorization.

It is the fourth factor in biological intelligence which poses special difficulties in finding its neural correlates. The *C* factor, or organized experience of the individual, is the memory factor in intelligence. It constitutes the organized framework of the "familiar" against which "new" experience is tested. It may be disturbed by a blow to the head which produces total or partial amnesia. It may also be disturbed by increased intracranial pressure or with advanced age. The selective action of traumatic amnesias, differential effects of hypnotic inhibition, and the selective impairments of experimental anoxia in normal subjects support a view of the *C* factor as comprising three types of memory: immediate, intermediate, and remote.

It is possible that immediate memory is a function of reverberating neural circuits activated by a perceiving neural network. But we must remember that such memory is normally of brief duration. Under optimal conditions of viewing motion pictures, for example, motion constancy breaks down for most normal subjects outside the limits of about twenty to thirty frames per second. If clear memory for each frame persisted as long as one second, the motion picture as we know it would be perceptually intolerable. However, persistence of immediate memory trace for tones and click stimuli has variously been reported from 2 to 12 seconds.

THE *A* FACTOR

If we apply a simple category test which requires the ordering of responses around such principles as size, brightness, position, number, color, to individuals with focal brain lesions and to a control group of normal individuals, striking quantitative differences emerge. These differences are shown in Table I. Note that both frontal and non-frontal lobe injuries are differentiated from the normal or control group, although the former are more so.

TABLE I

AVERAGE SCORE ON CATEGORY TEST FOR CONTROL GROUP, FRONTAL LOBECTOMIES, AND NON-FRONTAL LOBECTOMIES

$N = 30$	$N = 28$	$N = 22$			
Controls Average X_1	Frontal Lobectomies Average X_2	Non-frontal Lobectomies Average X_3	Significance of Difference, P		
			X_1, X_2	X_1, X_3	X_2, X_3
37.22	94.50	72.30	$t = 8.03$ $P < 0.001$	$t = 2.78$ $P < 0.006$	$t = 1.20$ $P < 0.033$

If we analyze the recurrent location of the lesions which produce the greatest differentiation noted above, we find the areas blocked out in Figure 3. It will be recalled that these areas correspond fairly closely with those regions of the brain and of the frontal lobes in particular which undergo myelination and other developmental changes relatively late.

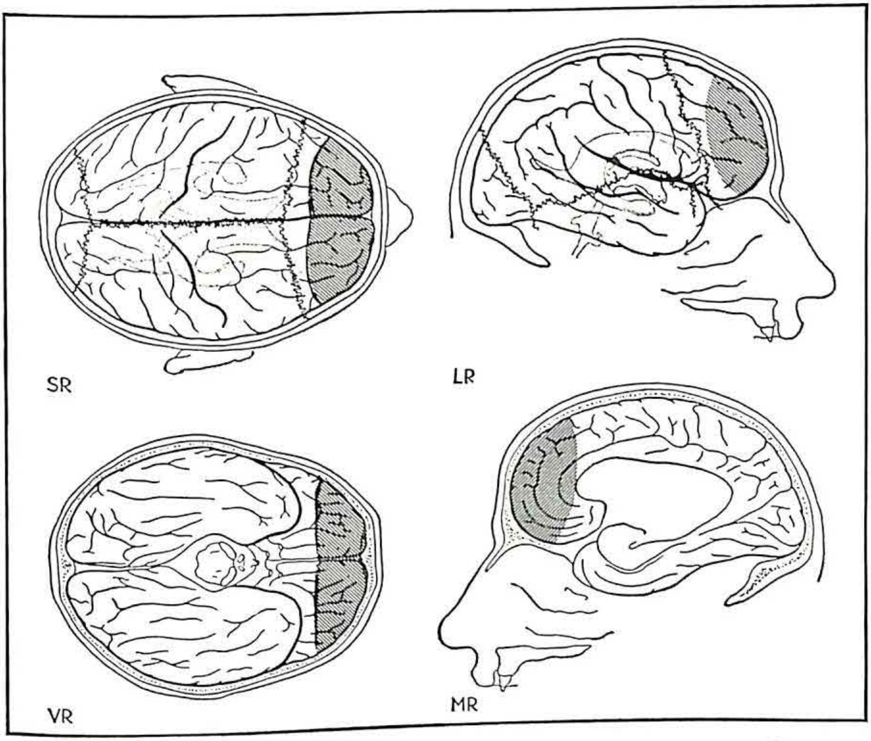


FIGURE 3. Showing the composite or common lesions in neurosurgical patients with greatest impairments of biological intelligence, reported by Halstead (12).

The category or grouping test as a whole consists of 336 items divided into nine subtests. These are presented serially by means of a projection apparatus. The modality or *D* factor considerations are kept to a minimum throughout by requiring differential response by means of four keyed switches. A chime and a buzzer instantaneously signal right and wrong choices. In this respect the test breaks with the customary pattern of thinking, for alas! no "bell" or "buzzer" signals the path of straight thinking to us normally. In other respects, however, the test does require the comprehension of essential and recurrent similarities in the presence of apparent differences, and vice versa. Since our earlier report we have had an opportunity to apply this test to a considerably larger sample population. This group now comprises approximately 300 individuals, distributed into six roughly equal subgroups: a control group of normal individuals, a special control group ostensibly recovered from a severe concussion, a group of severely neurotic patients, a group of chronically ill patients with such diseases as brucellosis and essential hypertension, a group with various endocrinological disorders such as myxedema, and a group with verified organic brain lesions or disease. How does the abstraction factor *A* stand up when examined in such individuals? The most convenient quantitative criterion for us is the relative number of errors each group makes. From nearly 200,000 responses, approximately 50,000 or one-fourth were errors from the standpoint of the test. If errors occurred on a chance basis, then each subgroup would be expected to contribute one-sixth of the errors or 16.7 per cent. Any significant deviation above or below this expectancy would presumably represent extra-chance causality.

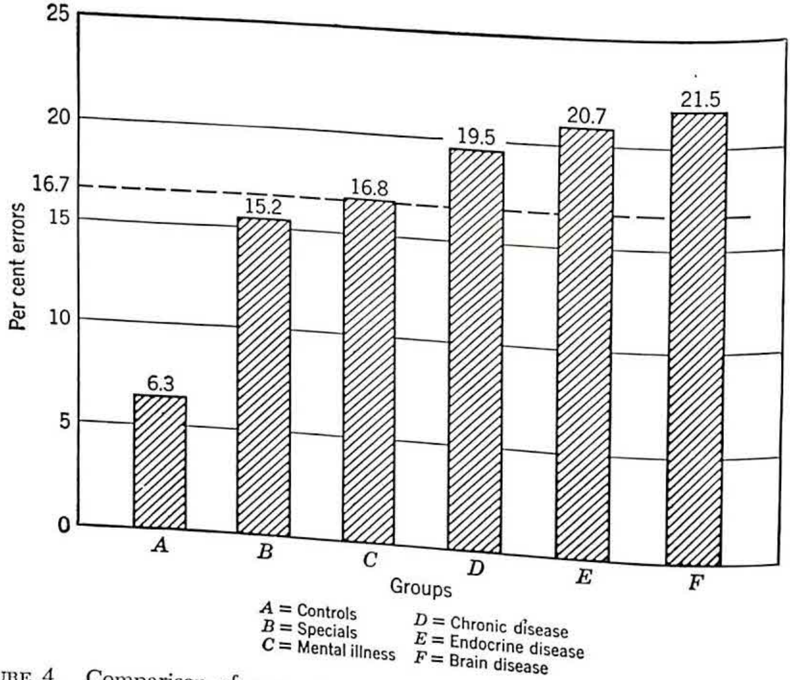


FIGURE 4. Comparison of percentage of errors made on category test by various diagnostic groups of patients. For explanation, see text; see Table II for distributions of errors by subtests.

In Figure 4 is shown the distribution of the errors made by the various subgroups. Note that the normal control group made but 6.3 per cent of the errors while the group with verified organic brain disease made 20.5 per cent, with the other groups falling in between. It is of interest that these intermediate groups behaved more like the known brain injury cases than like the normal control groups. That this difference is not attributable to the fact of hospitalization is safely indicated by the inclusion in our normal control group of orthopedic and obstetrical cases with comparable periods of hospitalization. Nor does the formal intelligence quotient account for the difference since none of the subgroups differs by the standard error of the psychometric test employed. Education background and economic station in life are adequately randomized in the subgroups so as to eliminate these from consideration. We appear to be forced, then, to turn to the nature of the category test to account for the obtained differences.

In the first place, the content of the test, although diversified, is simple. Very similar material has been made effective in the monkey (13). Is there something about the test which in effect asks a different question of the normal control group than of the other subgroups? This is, of course, the difficult question. Unfortunately we cannot answer it by asking the subjects directly. For verbally reported introspections on the abstracting process have, in our experience, proved to be thoroughly unreliable, not only for our severely impaired brain injury cases but for our normal individuals as well. This is probably no less true for the prestige introspections of men who have established eminent reputations on other grounds. It is the method of introspection which is inadequate. The language processes are rarely, if ever, directly coupled with the abstracting process. Abstraction is, of necessity, prelinguistic. It is for this reason that the aphasic patient may show no impairment of the abstracting process although he can verbalize no part of it.

We can, however, examine the ways in which the various subgroups distribute their errors on the nine subtests of the category test. This should give us some indication as to whether grossly different questions are involved. For example, it could be that the test is simply too long for all but the normal individuals and that “fatigue” or “boredom” intervenes to produce increasing arrays of errors. If we examine the actual percentages of errors made by the various subgroups on each of the subtests, as shown in Table II, we see that no very striking differences are apparent. The control individuals distribute their errors throughout the test much in the same way as the others. In fact, on the ninth or last subtest, where fatigue might be expected to be greatest, no true difference in percentage of errors is present for any of the sub-

TABLE II

Showing Distribution of Percentage of Errors on Category Test According to Diagnostic Groups

Category Subtests	C	Sp.	M.I.	C.D.	E.D.	O.B.D.
I	0	0.2	0.1	0	0	0.3
II	7.2	3.5	3.8	3.5	3.1	3.5
III	11.3	14.8	17.3	19.9	20.2	15.2
IV	17.1	14.4	15.6	15.2	15.0	14.8
V	15.3	20.6	15.3	15.7	20.1	16.7
VI	5.9	13.0	8.8	9.7	12.1	11.4
VII	21.4	14.3	17.3	14.5	10.9	15.1
VIII	12.7	9.4	12.3	10.8	9.5	12.8
IX	8.9	9.9	9.5	10.5	9.1	10.2

groups of individuals. Of course, the fact that it is the last subtest might induce goal expectancies which would counterbalance fatigue in this particular instance. But then we note that the control group made 21 per cent of its total errors on the seventh subtest in comparison with 15 per cent for the brain-damaged group. The evidence provided in Table II seems to indicate that much the same yardstick is being applied to the various groups of subjects. Wherein arises the differences in performance?

Perceptual difficulties cannot account for the failures since the subject is always able to describe the items in detail. Likewise, failure of brief memory seems to be ruled out by the performances on the last test which is a recognition test. Our evidence suggests that the differences arise in direct proportion as the task is blind for the various subjects. At various times the normal subjects seem to ignore very obvious dimensions of the stimulus configurations. They usually begin the test by isolating some stimulus dimension which is sufficiently recurrent as to produce temporary signs of successful performance. But when this initial cue proves to be but an incidental rather than a necessary element of the task, they may persist for several items in projecting their initial assumption onto the materials. In this respect they appear to differ only quantitatively from the brain-injured individuals; yet this difference is all important. They do shift more readily to other aspects of the situation and, hence, discover the necessary and sufficient attributes among the ambient dimensions of the stimuli at a much earlier stage. Ordering of their behavior with reference to test stimuli is at once more conscious and insightful, involving active effort. But lest we consider these latter qualities characteristic of the normal individual, let us examine another type of ordered behavior wherein the normal individual generalizes as blindly as does our most severely brain-injured individuals.

SCHEMATIC FACES AND AFFECTIVE BEHAVIOR

Some years ago Brunswick and Reiter (5) undertook an interesting study of certain physiognomic stereotypes in normal individuals. They employed ten judges. Using the method of paired comparisons, they asked their individuals to match up certain personality traits with schematic outlines of faces. Out of several hundred comparisons, they were able to isolate a dozen or more relatively strong schematic faces which were commonly associated with personality traits. It occurred to me to apply such materials to the study of human brain-injured individuals. Professor Brunswick kindly made available to me photographic negatives of his originals. From these I selected nine faces for purposes of my study (Halstead [11], Figure 212). Three of these faces had been found by Brunswick and Reiter and also by Samuels (26), working with Harvard students, to be strongly associated with such desirable personality traits as gayness, good character, intelligence, beauty, youth, et cetera. Five of these faces, on the other hand, had been found to be strongly associated with such undesirable personality traits as sadness, ugliness, bad character, unintelligence, and old age. The other face, J9, was found to be psychophysically neutral, being chosen with equal frequency for desirable and undesirable traits.

Over a period of several years we have had an opportunity to study the responses of several hundred individuals to the schematic faces presented to them by a modification of the method of limits. The extent to which ordered behavior of a certain type with reference to schematic faces occurs among our subjects is shown in Table III. Here the percentages of various subgroups who selected the schematic faces according to desirable (*D*) and undesirable (*U*) are shown for each of fourteen traits. Thus 86 per cent of our controls chose one of the expected three faces as being the most gay where chance would have yielded but 33.3 per cent. On the other hand, 95 per cent of them chose one of the expected five faces as being the most sad when chance would have yielded but 55.6 per cent. Note also that our brain-injured cases and our severely neurotic patients matched gayness and sadness in a very similar way. In fact, if we look at the various pairs of personality traits shown in this table, we are impressed by the striking similarities rather than the differences in the grouping behavior.

In this type of behavior, our normal individuals are just as irrational as our brain-injured individuals or as our psychiatric patients. It is difficult to see that such ordering behavior is on other than a blind basis. It seems quite unlikely that the child is born with an a priori notion that the distance between the eyes, the height of the forehead, the length of the nose, and the location of the mouth, the four varying elements in this series of faces, are invariable attributes of gayness, intelligence, et cetera. It is much more likely that the child quite unconsciously makes accidental associations with one or more of these elements. Since these associations are never brought directly into his consciousness, thus becoming subject to review in the face of contradictory evidence, generalization takes place after the first few experiences. From that time on he singles out for attention those associations through his daily experiences which match his now built-in expectancies. We are not yet clear when this process begins, but we have found one instance where a child of four years, ten months, required but slight translation of the trait names to show this conformity or stereotypy in thirteen of the fourteen judgments.

TABLE III

AFFECTIVE EQUIVALENCE IN GROUPING SCHEMATIC FACES ACCORDING TO PERSONALITY CHARACTERISTICS

Characteristic	Subjects					
	Controls $N = 102$		Brain Disease $N = 158$		Mental Illness $N = 103$	
	D	*U*	*D*	*U*	*D*	*U*
Gay	86		86		90	
Sad		95		92		87
Beautiful	92		92		80	
Ugly		92		91		93
Good character	83		77		80	
Bad character		76		81		76
Intelligent	68		77		73	
Unintelligent		89		82		84
Likable	86		86		84	
Unlikable		86		85		84
Young	90		87		91	
Old		89		86		89
Energy and determination	60		64		56	
Lacks energy and determination		86		86		85

Chance expectancy: $D = 33.3$ per cent; $U = 55.6$ per cent.
For deviations from chance: $P < 0.001$ or less.

This situation is not fundamentally different from the irrational or blind learning that takes place when a newcomer begins to take on the dialect of the community or in the process of identification of the child with one or both of his parents. Our groups of subjects are sufficiently heterogeneous to suggest that these irrationals are universals, at least so far as the western world is concerned. They are no less significant and statistically much more frequently encountered than instances of insightful or rational categorization. They comprise the social cement of society and, like the mason's mortar, they at once bind together and hold apart. I have not yet encountered a brain lesion severe enough in man, compatible with viability, to destroy these irrationals, nor have I failed to find them in all walks of life. It is this strong projective tendency to fix upon particular aspects, which we found to occur under the more restricted conditions of our category test. Herein lies a clue to the nature of the type of abstraction or grouping behavior that yields an insightful grasp of principles. The organism works initially less directly with the external material than with a priori expectancies. True categorization is achieved only when these expectancies have been put through a special set of operations in which a redistribution of affective loadings or valences takes place. The altered content must be rendered affectively equivalent. It is this type of work for which the organism is perhaps least prepared by its biological heritage. It requires deliberation with denial of immediate gratifications. It requires that the organism be able to protect its state of deliberation in the face of mounting frustration. If the organism is to maintain deliberativeness in the face of mounting frustration, there must be an adequate reserve of power, cortical or intellectual, for, as much experimental work indicates, frustration per se does not add power to the task but rather is parasitic on it.

FACTOR ANALYSES AND THE BASIC FUNCTIONS

Considerable help has been forthcoming in recent years from students of factor analysis in the identification of various attributes of intellect. The work of the late Charles Spearman of England and his associates and especially of Thurstone and his associates in this country has converged more and more on the concept of intellect as consisting of discrete but correlated functions. Several such functions or factors have been identified with reference to particular behavioral situations, but as yet the hierarchical relationship of these functions to other functions, including intellectual level (Binet), of the organism remains unknown. I have elsewhere reported factor analyses which support our interpretations of our evidence for the existence of the abstraction factor and the power factor in intellect. Additional factor analyses of the large bodies of data which we have subsequently carried out lend substantial support to our original interpretations. These are now completed and will be published at an early date. It is very probable that Spearman's general factor, *g*, which he finally interpreted, in his last paper published posthumously, as an abstraction factor (29), Thurstone's induction factor, *I* (32), and my *A* factor are the same factor. It is also quite probable that this is the same factor referred to by Goldstein (9) which he has designated in clinical terms as the abstract attitude. The power factor has not yet been identified as such by other investigators. The reason for this is that appropriate indicators for reflecting its variance have not yet been employed by them. This will doubtless come about in due time as the need for validation of postulated intellectual factors in terms of biological functions of the organism is recognized.

THE FRONTAL LOBES AND THE INTELLECT

We directed our attention at the outset to those parts of the brain which in terms of developmental criteria constitute the "new" brain in man. A considerable fraction of this "new" brain, we found, is in the anterior portions of the frontal lobes. From the results of applying a variety of indicators to frontal lobe and non-frontal lobe cases, we noted that the greatest impairment was obtained when areas of the "new" brain of the frontal lobes were directly involved (12). The clinical manifestations of damage to these areas in man indicate that these structures are intimately involved in the regulation of personality, but they do not specify the ways in which this regulation is brought about. Neither do they indicate to what extent the resulting alteration is primarily intellectual or cognitive in character as opposed to other functions.

It is true that the individual with even mild frontal lobe damage may have mild or severe losses in recent memory (the more intellectual the type of occupation, the earlier this symptom appears), difficulties in organizing and planning, coarsening of esthetic, intellectual, and moral judgments, a heightened tendency towards impulsivity, trigger-ish temper and grandiosity, insomnia without somnolence, and anxiety without insight. But these clinical traits or symptoms have never been adequately analyzed for common and unique factors.

The results for a variety of our experimentally controlled indicators indicate that damage to one or both prefrontal lobes is associated with several irreversible impairments, impairments which are detected when the formal psychometric I.Q. is normal or even superior. Functions mediated through vision, touch, and hearing are disturbed. Following either unilateral or bilateral prefrontal lobectomy, visual acuity and perimetric fields are unchanged, the audiogram is unchanged, and the cutaneous senses of the body and of the hands in particular, such as pain, pressure, temperature, and two-point limen, are unchanged. Nevertheless, certain tasks mediated through these modalities or processes are disturbed. Obviously these functions, and possibly others which we have not studied in comparable detail, must be disturbed at a higher level of neural organization. The evidence available indicates the anterior regions of the frontal lobes. It was this body of evidence which led me to a gradient theory of biological intelligence, the most differentiated aspect of which constitutes intellect (12).

Lashley (21) has marshalled an impressive array of evidence that the receptive areas of the cortex are themselves capable of processes of generalization "not fundamentally different from higher intellectual activities." But he is careful not to limit intellect to sensory processes alone. His evidence for the rat indicates that visual impressions consist of organized objects seen against a less coherent background. The figure-ground relation appears to be as basic for the sensory processes of the rat as for man. This relation is the first attribute of the stimulus to be differentiated and is as much a given element of the situation as any other attribute or dimension of the stimulus. Discriminative reactions in all modalities, when analyzed, are found to be based upon recurrent relational attributes of the stimulus. In fact, the initial stages of learning, both in man and in infrahuman forms, involve a time-consuming search for the necessary and sufficient relational cues as the basis for adaptive responses. This process of search usually does not include all of the possibilities in the situation even in the relatively simple material of our category test. In other words, some relations, often the effective ones, are ignored. The failure of some relational dimensions of the stimulus to register in consciousness accounts for the failure of the language to parallel directly the abstracting process both in our normal and pathological subjects. When asked why a particular response was made to hetero-dimensional stimuli involving color as the effective cue, the normal or pathological subject who has responded correctly may be unaware or may even deny that color was present. Similarly, such dimensions as number, position, brightness, size, shape, and spatial distribution may be ignored. The abstract process (*A* factor) involves active scanning and leveling or "landscaping" of the stimulus. Affective accents or valences associated a priori with the physical dimensions of the stimulus must be redistributed, some being raised here, others lowered there, until there is affective equivalence for the "relata."* This step requires vernier posturing of the organism with a high level of attention. The attentional field in this phase is broadly distributed and hence requires some leveling of the figure-ground relation characteristic of perceptual attention. It is as if the cortex were able to throw the central object slightly out of focus and then bring it back into clear consciousness. This step appears to be necessary for the effective relation to emerge in consciousness, where it gives up identity with a particular stimulus for inclusion in a group. It is in this sense that the relational attribute becomes the organizer for the category.

---

* Klüver's term for specifying the physical as opposed to the relational attributes.

It is unlikely that this process is a new one in nature. In fact, it would appear to bear some resemblance to events which may be basic to protoplasmic processes themselves (12).

Once the category is organized, its schema exerts an ordering influence on the succeeding situation and so on. The building of isomorphic schemata requires time, often much time. Hence the process is vulnerable to many sources of disturbance such as postural, attentional, or emotional, the effects of frustration. The process of perception is fundamentally similar, but is differentiated by the time dimensions of its initial organization in the outcome.

The two stages of perception and abstraction manifest themselves clearly in one of our indicators, a tactual form board test which we have found to have a substantial loading on our *A* factor. I have described this test elsewhere (11). The form board consists of ten cut-out wooden shapes which must be fitted into their appropriate recesses on a rectangular shaped board. The subject is never permitted to see this material. He is given an initial trial with the dominant hand and then a second trial with the other hand. After this, a third trial is given in which both hands are used. By the end of the first trial, the brain-damaged as well as the normal subjects can clearly identify the shape of each block. Yet they have no clear notion or schema for the board at this stage. Some learning has usually taken place with normal individuals, however, since they usually take less time for the second trial in spite of the shift of the task to the non-dominant hand. A considerable increment of learning occurs during this second trial as evidenced by further reduction of time in the third trial. When asked to draw an outline of the board, locating the shapes in their proper place, as they imagined the board to be, following the third trial, the normal individual shows that he has developed a good map or cortical schema of the stimulus. This is not the case in those individuals in whom the prefrontal lobes of the brain have been removed. This is shown clearly in the drawing of subject J. K.,* a bilateral prefrontal

---

* Referred to us for study by Dr. Spafford Ackerly, University of Louisville.

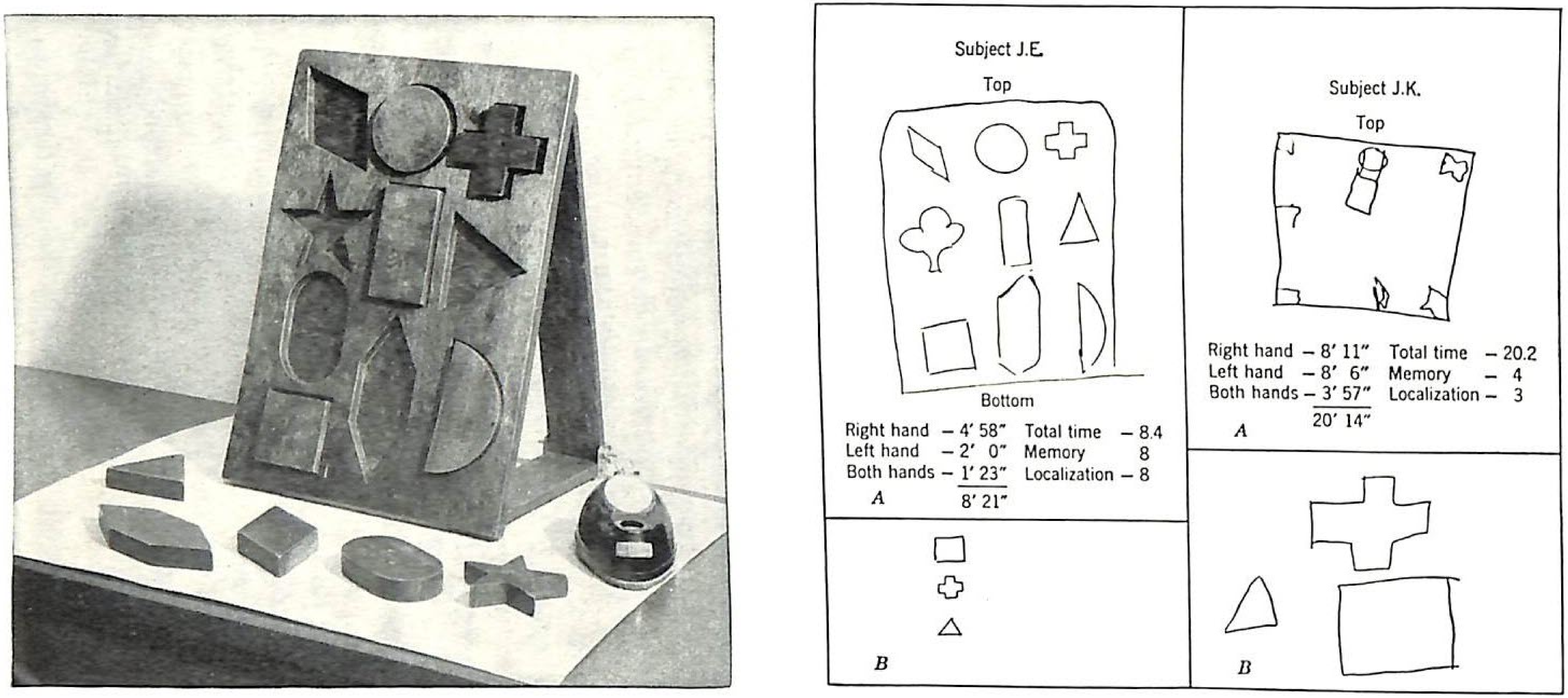


FIGURE 5. *Left:* Seguin-Goddard form board, which is solved without aid of vision. *Right:* comparable diagrams of board made from memory by control subject (left side) and bilateral frontal lobectomy (right side). Both subjects have normal I.Q.'s. Relative abilities to draw to instruction are shown in *B* in performance items from aphasia examination. For explanation, see text. (After W. Halstead, *Brain and Intelligence.* Chicago: University of Chicago Press, 1947. By permission of the author.)

lobectomy, in comparison with subject J. E., a normal control (Figure 5*A*). These individuals have the same formal psychometric I.Q. Note that the differences do not arise from the inability of either subject to draw a square, cross, or triangle, since they did this as part of an aphasia test (Figure 5*B*). Each of these three shapes is involved in the tactual form board situation. Subject J. K. had 250 per cent more time in contact with the test materials than subject J. E. Yet her drawing indicates that by comparison, the resulting schema is fragmentary and non-isomorphic, both in its content and in its spatial structure.

Its incorporation into the central integrated field factor, *C*, provides a built-in distortion which casts its influence into the future ordering of related behavior. This distortion is no less marked than nor fundamentally different in kind from that which characterizes the performances on our category test of the mentally ill subjects. They project non-isomorphic schemata upon the stimulus relata in the same way that the patients with verified brain injury do. Cascading of such distortions may account for much of their general state of maladjustment. They do not learn in terms of true categories but rather in terms of pseudocategories or partials. Such irrational learning is blind learning. As we noted earlier, this tendency towards irrational learning is all too strong in the normal individuals. In an open situation unaccented by focal rewards or punishments, as exemplified in our schematic face situation, the order of irrational learning encountered in our normal control individuals, our patients with known brain damage, and our psychiatric patients is virtually identical. This fact would appear to raise some doubt that studies of irrationals can provide the avenue to basic understanding of mental illness. In defining the parameters of healthy biological intelligence, the evidence which we have examined indicates that the cognitive processes and their associated rational learning towards insightful outcome hold the key to man's distinctive adaptive capacities in the behavioral universe. Our evidence indicates that biological intelligence is organized in terms of isomorphic schemata from the past, an adequate *A* factor supported by sufficient power (*P*) for exploring the present, and appropriately differentiated inward and outward avenues of exteriorization (*D*) for communication. The system is modulated both from within and without, in terms of its experiences on the one hand and the exigencies at the periphery on the other—a biological system with sufficient degrees of freedom to face the "future" with the capacity for controlled adaptation and without undue anxiety.

What are the necessary and sufficient mechanisms in the brain which bring these events to actuality? Here we are deprived of relata and must carry out our abstractions in terms of postulated relations. It is here that our model building helps, provided there is any hope of achieving a model with sufficient relational structure to the brain. Some investigators will take heart from the observations of Professor Bartelmez to the effect that "so little is known about the particular functions of the brain that no speculation can be said to be foolish." With the absence of relata we can be sure that model builders will design in terms of their expectancies or personalized schemata. From the seventeenth century on we have had numerous examples of mechanical, electrical, hydraulic, electromagnetic, and, more recently, electronic models seriously proposed as the analogue of brain function. Yet one looks in vain for a chemical model of brain processes. Some time ago I induced a friend in biochemistry* to undertake with me the task of setting up a chemical model for mental functions.

PROTEIN ORGANIZATION AND MENTAL FUNCTION

It seems that certain relationships to memory and to rational and irrational forms of learning become possible if we assume that in rendering nucleoproteins functional as a result of individual experience, an essential act is the transformation of proteins from random to oriented, organized configurations. In line with current concepts of protein structure and properties, proteins may have fully extended chains, the so-called fibrous protein. The protein chains can, however, form intermolecular hydrogen bonds, and, depending upon their amino-acid composition, may form a folded structure. The globular proteins are considered to consist of fully folded chains. The relationship between fully folded and fully extended configuration of some proteins appears to be related to their biological activity (30). Protein molecules, by virtue of hydrogen bonding and resonance stabilization, can form lattice structures, that is, repeating periodic structures which have to greater or less degree the properties of a crystal (25). The current picture of the genetic mechanism is an illustration of these ideas of protein structure applied to a biological problem. The gene is conceived of as a protein, probably nucleoprotein, which has a highly oriented, specific structure. In the cell economy, the gene acts as a template on which replica molecules are formed (2). In a very real sense the genetic apparatus is a memory (recapitulation) device. If, in place of endowed template molecules of the chains, we postulate the formation of templates as a result of individual experience, then some of the present knowledge of chemical genetics may be applicable directly to problems of mental function. From this point of view, the cell of the central nervous system and the germ plasm have this in common, that an essential aspect of their activity depends on the presence of template molecules. But in the case of the brain not all of the templates are an endowment but rather some arise from external stimuli. Organized protein structures are considered to have electrical conducting powers which are lacking in random aggregations of protein molecules.

---

* Dr. Joseph Katz of the Argonne National Laboratories in Chicago.

Szent-Györgyi (30) has recently applied such concepts of protein organization to the problem of muscle contraction. In accounting for electrical conductivity, he postulates that the "actual electrons of the protein particle form one common system belonging to the whole particle, in analogy to the modern theory of solids in which electrons belong to the whole system and cannot be correlated to single atoms," and that common conduction bonds extend over a great number of protein molecules. It is as if in the ordering or organization of protein chains, the outer electrons are abstracted from the protein particle to form a generalized conducting system.

As to where the ordering of protein molecules takes place in the brain, several possibilites arise, including various components of the cell and its processes. It seems likely that nucleoproteins are involved since the ability to act as a template seems to be fairly well restricted to these substances. The possibility of a direct relationship between the ordering of protein molecules and certain mental processes arises in the observation of Hydén and Hartelius (16) that the protein-producing system is poorly developed in psychically disordered persons, and that, in certain ganglion cells from the cerebral cortex of the frontal lobes of persons suffering from psychic disorders, the amount of polynucleotides and proteins is markedly decreased as compared to those of healthy individuals.

REFERENCES

1. Bailey, P. Concerning the organization of the cerebral cortex. *Texas Reports Biol. Med.*, 1948, 6, 34–56.
2. Beadle, G. W. Biochemical genetics. *Chem. Rev.*, 1945, 37, 15–96.
3. Binet, A., and Simon, T. *The intelligence of the feeble-minded.* Baltimore: Williams & Wilkins Co., 1916.
4. Breasted, J. H. *The Edwin Smith surgical papyrus.* Chicago: University of Chicago Press, 1930.
5. Brunswick, E., and Reiter, L. Eindruckscharactere schematisierter Gesichter. *Ztschr. f. Psychol.*, 1937, 142, 67–134.
6. Caspersson, T. The protein metabolism of the cell. *Naturwissenschaften*, 1941, 29, 33–43.

7. Conel, J. L. *The postnatal development of the human cerebral cortex.* Cambridge, Mass.: Harvard University Press. 3 vols. Vol. I, 1939; Vol. II, 1941; Vol. III, 1947.
8. Flechsig, P. *Gehirn und Seele.* Leipzig: Veit & Co., 1896.
9. Goldstein, K. Clinical and theoretic aspects of lesions of the frontal lobes. *Arch. Neurol. Psychiat.*, 1939, **41**, 865–867.
10. Halstead, W. C. A power factor (*P*) in general intelligence: the effects of brain injuries. *J. Psychol.*, 1945, **20**, 57–64.
11. Halstead, W. C. Brain injuries and the higher levels of consciousness. Chapter XX in: *Trauma of the central nervous system. A. Research Nerv. & Ment. Dis., Proceedings*, 1943. Baltimore: Williams & Wilkins, 1945.
12. Halstead, W. C. *Brain and intelligence.* Chicago: University of Chicago Press, 1947.
13. Harlow, H. F. Solution by rhesus monkeys of a problem involving the Weigl principle using the matching-from-sample method. *J. Comp. Psychol.*, 1943, **36**, 217–227.
14. Hebb, D. O. The effect of early and late brain injury upon test scores and the nature of normal adult intelligence. *Proc. Am. Philosophical Soc.*, 1942, **85**, 275–292.
15. Hebb, D. O. Man's frontal lobes. *Arch. Neurol. & Psychiat.*, 1945, **54**, 10–24.
16. Hydén, H., and Hartelius, H. Stimulation of the nucleoprotein production in the nerve cell by malononitrile, and its effect on psychic functions in mental disorder. *Acta Psychiatrica et Neurologica*, Suppl. XLVII, 1948.
17. Jakob, C. El lobulo frontal. *Folia Neurobiol. Argentina*, 1943, **3**, 1–141.
18. Kappers, C. U. A., Huber, G. C., and Crosby, E. C. *The comparative anatomy of the nervous system of vertebrates, including man.* New York: The Macmillan Co., 1936. Vol. II.
19. Köhler, W. *The mentality of apes.* New York: Harcourt, Brace & Co., 1925.
20. Lashley, K. S. *Brain mechanisms and intelligence.* Chicago: University of Chicago Press, 1929.
21. Lashley, K. S. The problem of cerebral organization in vision. *Biological Symposia*, VII, Ed., H. Klüver. Lancaster: Jacques Cattell Press, 1942.
22. Lashley, K. S., and Wade, M. The Pavlovian theory of generalization. *Psychol. Rev.*, 1946, **53**, 72–87.
23. McCulloch, W. S. Modes of functional organization of the cerebral cortex. *Federation Proceedings*, 1947, **6**, 448–452.
24. Nielsen, J. M. *Agnosia, apraxia, aphasia.* New York: Paul B. Hoeber, Inc., 1946. 2nd ed.
25. Pauling, L. Theory of the process and formation of antibodies. *J. Am. Chem. Soc.*, 1940, **62**, 2643–2657.
26. Samuels, M. R. Judgments of faces. *Char. & Pers.*, 1939, **8**, 18–27.
27. Spearman, C. The proof and measurement of association between two things. *Am. J. Psychol.*, 1904, **15**, 72–101.
28. Spearman, C. *The abilities of man.* New York: The Macmillan Co., 1927.
29. Spearman, C. Theory of general factor. *Brit. J. Psychol.*, 1946, **36**, 117–131.
30. Szent-Györgyi, A. *Nature of life.* New York: Academic Press, 1948.
31. Thurstone, L. L. Multiple factor analysis. *Psychol. Rev.*, 1931, **38**, 406–427.
32. Thurstone, L. L. *Multiple-factor analysis.* Chicago: University of Chicago Press, 1947.
33. Watson, J. B. *Animal education.* Chicago: University of Chicago Press, 1903.
34. Wiener, N. *Cybernetics.* New York: John Wiley and Sons, 1948.

35. Woolsey, C. N. Patterns of sensory representation in the cerebral cortex. *Federation Proceedings*, 1947, 6, 437–441.

36. Yerkes, R. M. The intelligence of earthworms. *J. Animal Behavior*, 1912, 2, 332–352.

DISCUSSION

DR. LASHLEY: Dr. Halstead's work represents what is almost the first attempt to explore in a systematic way a great variety of possible defects arising from localized injury in the brain. He has been engaged for a number of years in devising tests for almost every imaginable function and trying them as to their effectiveness in revealing defects. This seems to me to be the best approach that we can make to the study of such things as frontal lobe deficiencies, aphasias, and the like. Earlier studies depended pretty largely on chance observations of defects, without systematic investigation to determine what functions may be correlated in the defects produced by localized brain injury. This then, I think, is the most promising method of approach to the whole problem of cerebral localization that has been made.

I would like to ask Dr. Halstead two questions with specific reference to the study of the frontal lobes. First, whether in a series of patients, he has cases which would be crucial for distinguishing between the effect of removal of tissue and the effect of malfunctioning of the remaining tissue. In our work with animals, which I said is not wholly transferable to man, we have found relatively few deficiencies from large lesions in the associative areas. I have come to feel that perhaps many of the symptoms which occur in man are the result of malfunctioning of scar tissue, of cells which are caught in an abnormal environment, rather than of the specific loss of cortical tissue.

The second question: In surveying Dr. Halstead's series of tests, I felt that those tests which are specific for the frontal lobes, the tests which reveal defects only as a result of frontal lobe lesion, all seem to involve some aspect of visual function. Even the form board which is used with blindfolded subjects requires perhaps the translation of tactile into visual space. We have found in work with monkeys that the combination of lesions involving the frontal eye-fields and the more anterior part of the cortex, not the frontal eye-field alone, seemed to produce defects which I am inclined to interpret as defects of visual attention rather than as primary hemianopia as interpreted by Kennard. It is a temporary condition, but it may persist for a considerable period. I raise the question whether the defects which differentiate the frontal lobe cases are really defects of intellect or merely some obscure disturbance of visual functions.

DR. HALSTEAD: Professor Lashley has the obvious advantage of histological access to his material. Many of *these* cases are still living, and we simply do not know to what extent there is pathological tissue. However, on a statistical basis, we would expect such pathology to occur with equal frequency among our non-frontal cases. If that were the basis for the differences in behavior, I would expect our occipital lobe cases and our parietal and non-dominant temporal cases to show an equal incidence of such disturbances. This is not consistent with our findings in fact. Our series includes one instance of removal of what was probably an essentially normal right frontal lobe for an unverified aneurysm, in a 35-year-old individual. Post-operative quantitative signs of impairment are as marked as in our operated brain tumor and scar cases. This would seem to approximate a crucial case.

Dr. Lashley raises the question concerning the extent to which the basic defect in our frontal lobe cases is essentially visual rather than intellectual. The distinction is an extremely complicated one methodologically. It is true that the visual hypothesis appears to fit a considerable part of our data. However, two of the tests which differentiate our frontal lobe cases involve auditory discriminations. Unfortunately, comparable data for smell and taste are lacking. The problem is further complicated by the failure in our experience of frontal lobe lesions to disturb such obscure aspects of visual function as the ability to relate physiognomic cues to personality trait names. It may be significant that Lowenfeld found it necessary to distinguish between a "haptic" and a "visual" type in the congenitally blind. It seems clear that frontal lobectomy does not produce the abolition of any function in the sense that occipital lobectomy yields a total and permanent sensory deficit in the corresponding hemianopic visual field. Rather, there appears to be a diminution in the elaborations of visual, tactile, and auditory functions and an associated impairment of intellect. The form of biological intelligence is unchanged, but the amplitude is diminished. I strongly suspect that the basic difficulty arises at the level of energetics, or what I have termed the power factor. I have observed the same order of impairment to be reversible under focal management in such non-neurological cases as pheochromocytomas and myxedemas. The recovery in such cases is selective, thus affirming the identity of my factors, and is associated with the return of intellectual ability.

DR. MC CULLOCH: I usually make it a habit to talk to Dr. Halstead about the things that worry me, because he is near enough. Therefore, I have very little to ask now. I don't think anybody realizes, without knowing him, the enormous number of tests that he has tried, done, and discarded. I think what is most amazing is actually hidden by his revealing to you what do come out as the positive findings. To me the amazing part of his work is the enormous mass of performances on tests which are not affected by these injuries to the brain. I wish he would say a word on that score, because I think it would be really helpful.

DR. HALSTEAD: It is true, I have groped and roamed over rather a wide range in trying to find the types of situations that would present special problems for organic brain injury—paper and pencil tests, interviews, moving picture situations, social environment, and so forth. I have not literally lived with these cases, but I have investigated their homes and have attempted to find out whether our laboratory findings would follow into the cultural situation. I will not feel that I have done a proper job until I can more adequately describe the non-equivalent situation because I think that therein lies the important clue for building an adequate battery of indicators. I hope, sometime, to put this material into orderly form so that we can see what the common factors are in the non-equivalent situations.

DR. KLÜVER: It is very gratifying that Dr. Halstead does not confine himself to using the tools of experimental psychology for the study of neurological problems only and that he has joined the internist in extending his analysis to other disorders. There are indications that the significance of nervous factors in various diseases is becoming more generally recognized by investigators in the field of internal medicine. In fact, Speransky has devoted a whole book to the role of the nervous system in the genesis of pathological processes. There is no doubt that experimental psychology may, in some instances, furnish indicators which are more sensitive and more precise than other indicators, and that such indicators may be utilized in detecting changes when other methods of investigation fail.

I should like to ask only two questions. First, I am wondering how far you want to go in attributing a functional significance to the myelogenetic data supplied by Flechsig and others. Available data on myelogenesis are based on the application of certain staining techniques, that is, on the occurrence of chemical processes and reactions the nature of which remains obscure. It seems that a myelination of fiber systems as determined by such techniques does not necessarily mean that the chemistry of the myelin sheaths undergoes no further changes in the course of development. For instance, Weil's data on the chemical growth of the brain of the white rat indicate that changes in the phospholipid content and in the amounts of the different phospholipid fractions occur at least until the age of 180 days, that is, until an age which corresponds to about 15 years in man. I do not know of any comparable studies on the human brain.

Second, Dr. Halstead's intensive analysis has thrown new light on the functional significance of the frontal lobes. A detailed discussion of his experimental findings would take us too far afield, but his emphasis on the relation of the frontal lobes to intellect raises an interesting question. If we consider the history of previous attempts to relate different portions of the brain to higher psychological functions and to the complexities of human personality, we find that investigators at one time or another have assigned the chief role to the frontal lobe or to the temporal lobe or to the parietal lobe. A neuropsychiatrist returning from a visit to scientific institutions in Europe before the war told me that the Psychiatric Clinic of the University of Vienna should really be called a Posterior Parietal Lobe Institute. It seems that the occipital lobe alone has escaped since no one has ever seriously maintained that it is of fundamental significance for the structure of human personality. Why this exceptional "purity" of the occipital lobe? Perhaps the data of physical anthropology are of some interest in connection with the problem whether there is a migration of functions towards the frontal pole (von Monakow's *Wanderung der Funktion nach dem Frontalende*). In studying the phylogenetic transformation of the human skull by comparing anthropoids, hominids, and man, the late Weidenreich reached the conclusion that it is the bulging in the parietal and posterior temporal regions which is characteristic of the last evolutionary stage. The question remains why the evidence obtained in various researches concerned with the role of the different lobes in higher functions and personality could be interpreted in such a way that outstanding investigators during the last fifty years implicated the frontal, the parietal, or the temporal, but not the occipital lobe.

DR. HALSTEAD: I feel that it is much too early to know how far we can go in relating the train of events underlying the myelination process to function. One of the difficulties involved concerns the level of the psychological question which can be asked for purposes of correlation. For example, Watson found that his 24-day rats with only 20 per cent of their fibers myelinated could answer successfully the psychological questions which he could put to his adult rats. But the analogue of the questions asked would by extrapolation correspond to a mental age of about two years in the human being, a stage of development in which intellect is not notably conspicuous. I have found some 12-to-15-year-old children with I.Q.'s in the neighborhood of 100 to do as well on my tests as highly educated adults, including college professors. Younger children, on the other hand, have considerable difficulty. This suggests to me that some maturation process, possibly including myelination, must reach a certain stage for the functions involved in my indicators to appear full blown.

Dr. Klüver raises the interesting question concerning the historical sparing of the occipital lobes from implication in the higher mental processes. Paralysis and blindness are among the more dramatic neurological defects. It perhaps is not surprising that they came under systematic investigation relatively early. Following the lead of Flourens, Panizza, and Hitzig, Munk and Schäfer had pretty well established the occipital cortex as a trophic center for visual functions considerably before Binet even began his work on the nature of feeble-mindedness. Thus accidental factors may have played a part.

However, I do not believe that this is the whole story. There is a certain precision and restriction in the defects which result from occipital lobe lesions in man which seems to set this lobe apart. What the occipital lobe patient can *see* he can integrate. The question of intellectual impairment as such somehow does not arise in the same sense that it does, for example, in frontal lobe defects where the patient somehow fails to *integrate* that which he can see, touch, and hear.

DR. NIELSEN: It was my great privilege in Atlantic City to go over this subject with Dr. Halstead over a bottle of ale, and whether that facilitated our understanding, I don't know. However, I think that we had a pretty good understanding. Dr. Halstead is the only psychologist that I have ever heard of who can tell by his psychological tests that the frontal lobes have been taken off. We have worked in a clinical way for a long time and, of course, have had to arrive at our own clinical interpretations, but Dr. Halstead is now showing us why we have been getting our interpretations. There are many cases, in the literature, and we have seen several, in which the frontal lobes have either been amputated or completely put out of function by tumor, and we have gradually learned how to tell that that is the case by clinical examination of the patient. If I may take just a couple of minutes to recite one case, you will see how evident it is.

It is the case of an automobile dealer who had a great reputation for veracity and reliability. He was, in fact, so reliable that you could take him your car, tell him to sell it and give you a new one—and you needed no receipt or papers of any kind. Then he began to be ill. First he went into an emotional depression which was interpreted by me, erroneously, as simply an emotional depression. I advised a vacation of a month, which he took, and he recovered from that depression. Then his wife told us that he was losing his agency, that the company had investigated and found great irregularities. He had taken in cars and had spent the money and given no account of it, and he had even denied receiving the cars at all. Not only that, he would get up at five o'clock in the morning, go to his place of business, returning at seven. When he was asked why he was not at work, he said that there was nothing going on over there and nobody was there anyway so what was the use of his being there. He would sit all day and listen to the radio—something that he had never indulged in before. He would sometimes go over to his place of business in the evening, again returning to say that there was nobody there to stay for. When his wife asked him about the payroll, he said, "Oh, they'll take care of themselves, they'll pay their own wages." He was completely bankrupt by the time of the investigation. His wife brought him to us again then, and I was able to say on the basis of his actions that he had a bilateral tumor of the frontal lobes. There was a complete change in his personality, and by then there was a little pressure shown in the eye grounds by which I was able to say tumor and not some other form of destruction. At operation he showed a bilateral tumor of the frontal lobes. We came to the conclusion, after a great deal of discussion, that the portion of the brain which is tested in the I.Q. tests is the portion from area 6 back. The portion from area 6 forward, that is, the prefrontal area, is the area, not for the storage of memory but for the knowledge of what to do with memory. Therefore, we have stated it very simply in this manner. We have two kinds of knowledge: the simple cognitive knowledge—the accumulation of facts; and the other kind—the knowledge of what to do with facts. The latter is stored in the frontal lobes. From area 6 back is factual knowledge; from area 6 forward is wisdom. And when a man loses his wisdom, there is something wrong with his frontal lobes.

DR. LINDSLEY: For a long time I have been convinced that in experimental psychology and in physiological psychology, we already have techniques which could be adapted from their original purpose and applied in this way, and I am sure that the stimulation of Dr. Halstead's work will direct a number of psychologists into this kind of application. I would like to emphasize, though, that in addition to the clinical, neuropathological, physiopathological human material, we can also seek the biological-physiological basis and possibly the morphological basis of certain psychological processes through other lines of attack. I am sure that Dr. Halstead will agree that these should likewise be followed up and correlated with such results as his. I have reference particularly to the phylogenetic or comparative type of developmental picture in animals, and to the ontogenetic or developmental picture in man. In this connection, and also in connection with his remarks about myelination of particular areas, I would

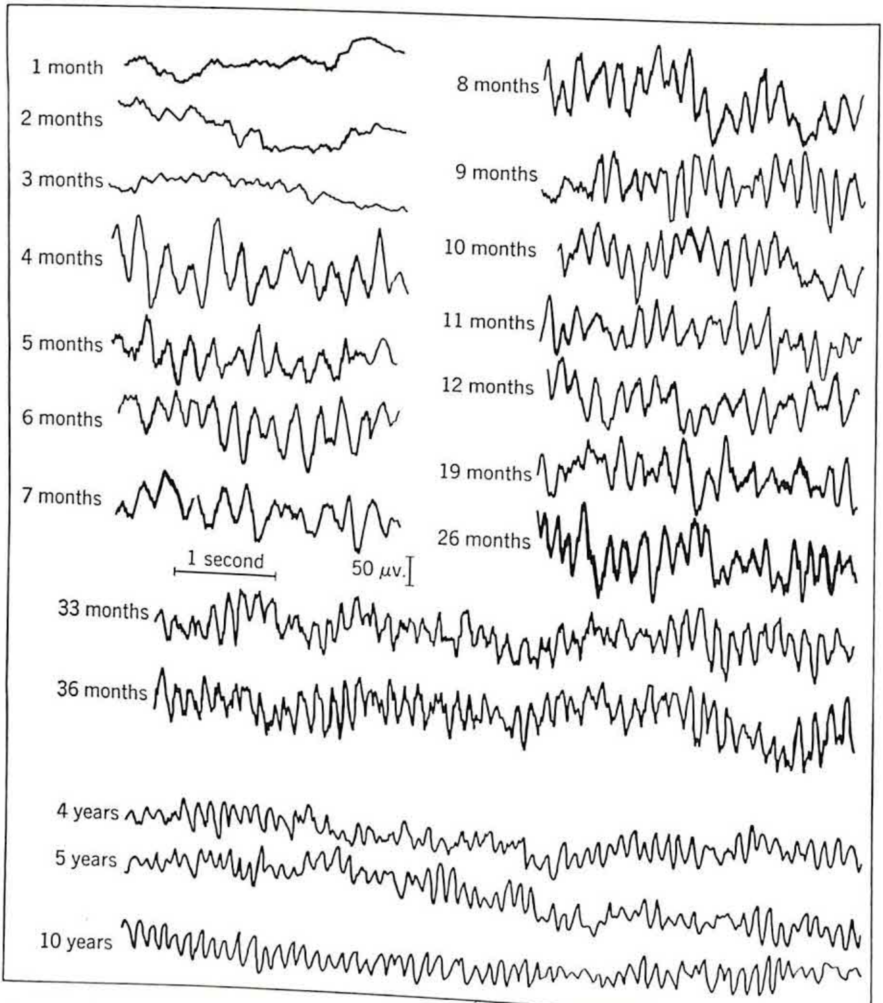


FIGURE 1. Development of the occipital alpha rhythm in a boy from birth to ten years. Note the onset of the rhythm at four months and the increase thereafter with age.

like to take just a moment, if I may, to show one slide which I think, in part, may confirm his implication about differentiation of areas in the cortex. I have done the same thing that Dr. Halstead has done in referring to some of Flechsig's and Conel's work in trying to relate some of the genetic developments in the cortex to what we know, at least, of architectonics and myelination. This slide was obtained from my own boy, who was born at a propitious moment in 1936, when I was starting to record some of these waves.

The records were taken from the occipital area. For the first three months, although there are evidences of activity, there are no well-developed or uniform patterns. Between 2 and 4 months and thereafter persistent rhythms develop. At that age the rhythm is 3 per second; it increases to about 6 per second at the end of the first year, and from then on rises more slowly until at 10 or 12 years of age it has reached adult frequency. Dr. J. R. Smith (who has done similar work on newborn infants) and I have both referred to the possibility that the development in the occipital area, as shown in the work of Flechsig and, more recently, of Conel, may bear a gross correlation with this general type of onset. This is, I believe, in substantial agreement with Dr. Halstead's remark about myelinization.

. . . .

DR. VAN HARREVELD: I am afraid that I won't be able to contribute anything to the main theme of the symposium on brain mechanisms in behavior. However I would like to make a remark which may not be too irrelevant. During the first two days of this symposium the reverberating circuit was emphasized. Many things, from facilitation to memory, have been explained on the basis of such circuits. The concept of the reverberating circuit is an unusually interesting and important one, and it is one of the great accomplishments of Dr. Lorente de Nó, not only to have shown that reverberating circuits are anatomically possible, but to have demonstrated, in a great number of ingenious experiments, that the reverberating circuit is an important factor in central nervous facilitation. However, it shouldn't be forgotten that there are other ways to influence synaptic conduction. One obvious way is the application of drugs which either depress or facilitate conduction in neural chains. Drugs can also uncover connections which are not functional under normal circumstances. It is well known that strychnine plays havoc with reciprocal innervation; motor neurons become accessible to stimulation, which before the application of strychnine could not be reached. There are present in the central nervous system other neural mechanisms which are similarly affected by strychnine. I am thinking of the synapse which connects the first and second sensory neurons. In these structures also connections can be uncovered by strychnine which are not accessible in unpoisoned animals.

Dusser de Barenne found that by local application of strychnine to the spinal cord a hyperalgesia can be elicited in the relevant dermatomes. When one repeats this experiment, one finds that the slightest touch of a hair on the skin may cause a reaction of the animal which indicates pain. An obvious explanation of this observation is that there exists in the spinal cord a connection between the tracts for touch and for pain which is normally afunctional but which can be made patent after the application of strychnine. Obviously, this hyperalgesia is present only as long as the strychnine effect lasts. We found a number of years ago that, by asphyxiation of the spinal cord for an appropriate period of time (around 25 minutes), it is possible to change the spinal cord permanently, in such a way that touch is apparently experienced by the animal as pain. After such an asphyxiation of the cord there develops in the ensuing 48 hours a hyperalgesia in the hind legs which is very similar to that described by Dusser de Barenne after local application of strychnine. It again seems likely that the impulses from the tracts in the cord for touch now spill over through unmasked connections into those for pain. This condition seems to be permanent.

Now, isn't what Dr. Liddell does when he develops in his sheep and goats conditioned reflexes, of the same order as what we do with a kind of sledge hammer procedure in the spinal cord? Doesn't he uncover connections which were there before, but were afunctional? The impulses produced by the buzzers and whistles can now reach effector neurons, either secretory or motor, which before were not available to these impulses. I feel a perhaps superficial resemblance in the very crude way of permanently uncovering existing connections by asphyxiation and the much more refined method that is used in the development of conditioned reflexes. The only possible importance of this comparison may be that it sets forth the hope that, in the future, we may be able to understand the mechanisms which are instrumental in unmasking existing connections during development of conditioned reflexes.

DR. LASHLEY: I would like to urge caution in the interpretation of pharmacological data and their use in explanation of behavior. After Dusser reported the spread of excitation or reduction of synaptic resistance as an effect of strychninization, I set up an extensive experiment to test the effect of this spread upon the rate of learning. I used both strychnine and caffeine, since the two drugs were assumed to have similar pharmacological action. Under strychnine, rats learned twice as rapidly as the normal controls; under caffeine, learning was greatly retarded. The results were statistically reliable and uninterpretable in terms of the supposed action of the drugs.

I question whether the facts justify the assumption that there is an opening of connections between conduction paths for touch and pain. Hyperalgesia may follow injury in nerve trunks, spinal cord, or thalamus. The evidence for separate conduction for touch and pain is not conclusive. There is some indication that the same receptor may produce tactile or pain effects, according to the frequency and duration of its discharge. The hyperalgesia resulting from strychnine may be the result of changes in frequency within a conducting system rather than of transfer of excitation from one path to another.

DR. VAN HARREVELD: In the method of asphyxiation used to produce hyperalgesia the spinal cord only is asphyxiated. The periphery remains normally oxygenated, and it therefore seems likely that the impulses set up by peripheral stimulation are unchanged. Two mechanisms have been proposed for the signaling of painful stimulations. One is a high frequency discharge in a pathway normally used for another quality of sensation. However, the work of Adrian, McKeen Cattell, and Hoagland does not favor the existence of such a mechanism. The other is a discharge along a specific pain tract. If the latter mechanism of pain signaling is accepted, a connection between the tracts of pain and other qualities of sensation has to be assumed to explain the results of strychnine application and of asphyxiation.

DR. WEISS: I don't think that you really ought to go home without hearing, as a note of caution, about some of the unexpected things that have come up lately in our knowledge of the neuron. We have been talking about the neurons as if they were more or less stationary fixed things, and this concept has been somewhat shattered. I am going to give you briefly the results of the last five years of work. You can find the evidence in a paper by Weiss and Hiscoe in the *Journal of Experimental Zoology*, the April 1948 number, where all the material of these five years is summarized. The facts are these: The mature neuron, not only the growing neuron, is in a state of constant renewal. Protoplasm is constantly produced only from the soma of the cell, the nuclear territory, and it is moved into the axon at a rate of, roughly, two millimeters a day (for a particular nerve of the rat). This movement of the protoplasm in a seemingly solid column serves to replace, apparently, the decaying proteins and other protoplasmic systems which cannot, like lower molecular compounds, be synthesized locally, but have to come from a central production source. There is constant breakdown in the nerve fiber and constant renewal from the center. What does this imply for the problems we are here concerned with? First, if the geneticists are right, there is no divergent differentiation among the genic nuclear systems of the various cell types. On the other hand, the cytoplasm of a nerve cell is strictly neural protoplasm, different from that of other cell types. Therefore, there must occur a conversion of the common primordial genic compounds (for example, proteins), as they come out of the nucleus and react with specifically configurated neural templates in the neural cytoplasm. The cytoplasm would do a recasting and molding job on the mother substances coming from the nucleus. This agrees very well with some of the ideas which have been developed at this institution. Dr. Pauling's, for instance, and Dr. Sterling Emerson's views on the effects of antibodies in gene reproduction have a close resemblance to this. That means that if we change the templates in the cytoplasm, which itself is not reproductive, by any sort of specific external influence which can pass into the neuron, such as antibodies or sensitizing chemicals or other unknown influences, then we change not only the existing compounds in the nerve cell, but also the pattern of cytoplasmic renewal. We have, here, for the first time, a mechanism which could explain how an external influence could specifically alter the constitution of a nerve cell so that the resulting change would become self-perpetuating.

A second thing that we have to consider is that the amount of synthesis, and therewith the size of the cell, seems to vary with the actual size of the functional periphery with which it is connected. A disconnected neuron immediately reacts by reduced synthesis; the overloaded neuron reacts apparently (we are not quite sure yet) with increased synthesis. The cell body of a ganglion cell can shrink and swell by as much as 40 per cent. According to Hyden, changes in functional activity have similar effects. It is inconceivable that the fine micro-arrangements of terminal connections at the surface of the cell remain unaffected by this functional up and down of the cell bodies. We must conclude, therefore, that with changing functional relations and changing anatomical relations, the finer connections of the cell body will also suffer. Therefore, we have here another hint at the constant breaking and making of the connections at the finer synaptic level of the cell.

I present these two points because they demonstrate clearly that we are dealing, not with a system that is static and rigid, the way it appears in the pictures of microscopic anatomy, but one that is subject to constant change.

DR. WOODBURY: We have had several models of the nervous system so far, and I have here another. I would like to suggest it for certain types of extremely abnormal behavior. The models during this session have been primarily built up of the smaller units—the neuron, the fiber tracts, the nuclei, and the other smaller aspects of the cerebral physiology. Working as I do with experimental convulsions, I have had occasion to use another type of model, or analogue, or whatever you wish to call it, and that is of the brain as a whole, not subdivided into parts, but considered simply as a giant neuron. It is applicable primarily to problems of experimental seizures, but it has points not of homology, of course, but of a very close correspondence to a single neuron, except that the orders of magnitude of the constants are different.

The brain is a unit; it has excitability; it differs from the neuron in one important respect—no single direct current impulse of any reasonable magnitude or duration will excite it—it requires several impulses. The minimum number is not certain, but it is probably two or three at the very least, and they need to be separated by a finite interval. Too close a spacing, and you get no excitability; too long a spacing, and the excitation between impulses dies out and no convulsion ensues. There is a threshold, a value below which the brain will not go into convulsions. The threshold varies with time and intensity of the impulse, so that you get an ordinary time duration curve, or rather a time intensity curve. The shorter the duration of the total stimulus (and the total stimulus includes many impulses), the higher the intensity; the longer the duration, the less the intensity; until you attain a rheobase for sixty-cycle alternating current stimulation. The chronaxie is in the neighborhood of a tenth of a second, as compared with much smaller values for the single neurons. It shows accommodation, very strikingly so—at low frequencies the intensity of current required is much greater than at somewhat higher frequencies. It shows a lag in excitation very similar to that of the peripheral nerve at high frequencies. The only thing is, the time constants are strikingly different. There is a period of refractoriness following convulsions, and a recovery of excitability; after the convulsion, the excitability is rather slow in recovery. After a convulsion, plotting time against the extent of normal excitability, one obtains a curve that is considerably different in form from that of the individual neuron, but shows much the same type of characteristics.

The brain has one other thing that is also of interest. That is the ability to respond, by multiple convulsions, to a continuing stimulus, either electrical or chemical. When the stimulus is a chemical, a convulsing drug in the blood stream which lasts for several hours, the animal will have one convulsion after another at approximately five-minute intervals—a convulsion, a period of refractoriness, a convulsion period, etc. This is very similar to the single cell under continual direct current stimulation where it responds by a series of impulses, or a series of firings. The threshold and other characteristics can be moved around by appropriate treatment with electrolytes of one kind or the other. Remove the calcium, the threshold goes down; increase the sodium, the threshold goes up; increase the potassium in the extracellular fluid, the threshold goes down. All in all, one can draw a very close analogy between the brain as it responds to convulsive stimuli, and the single neuron as it responds to the excitatory stimuli. It makes a rather interesting model of the brain responding as a unit.

DR. WIERSMA: If I understood Dr. Woodbury, he made a statement, I think, that the chronaxie is about a tenth of a second. Is that correct?

DR. WOODBURY: I do not have with me the exact figure, but it is of that order of magnitude.

DR. WIERSMA: I would like to point out that in this respect you seem to be making an error similar to that made by several Frenchmen with regard to the crustacean nerve-muscle preparations. When chronaxies are determined for systems where repetitive impulses are necessary in order to obtain the reaction, one often obtains pseudochronaxie values. They will depend on the frequency of stimulation, and it is uncertain what their relation to the true chronaxie is, if any. We determined the true chronaxie of elements stimulated in order to obtain electronarcosis in dogs, keeping the necessity of repetitive stimulation in mind; and we found that, so determined, it was exactly that of single nervous elements, namely about one-tenth of a millisecond and not a tenth of a second.

DR. LIDDELL: I would like to make two comments. First, on your intriguing analogy, we can demonstrate refractoriness of this one-neuron brain in the sheep and goat. If the metronome signaling shock clicks, the sheep flexes at the clicking and then at the shock; if the metronome continues after the shock, his single-neuron brain becomes refractory for definitely predictable intervals before flexion of the leg is resumed. Second, may I reopen the record of Dr. van Harreveld's remarks, because I think there may be an unmasking factor operative in the experimental neuroses which become permanently established in our animals in consequence of difficult conditioning. The resemblance to Dr. van Harreveld's strychnine experiment is closer than I had realized, in that our animal, when these carefully timed afferent volleys accompanying signal and shock are continued for a time, develops a pattern of chronic agitation, but moreover develops something similar to a hyperalgesia of the trained forelimb. He will flinch if the limb is lightly touched. It looks very much as if there might be a permanent unmasking of delicate defensive mechanisms, the operation of which we cannot demonstrate in the normal animal.

DR. HALSTEAD: I would like to mention one observation which, it seems to me, might conceivably represent the case of a figural after-effect sustained by means of a reverberating circuit. If you rotate pigeons that are blindfolded you get a rather dramatic nystagmic pattern that involves the total head. It is very easy to record. Under conditions of repeated elicitation, the effect will undergo a marked reduction. However, the point that I wanted to direct your attention to is that after the primary postrotational nystagmus has run its course, and following a delay of about 6 to 10 seconds, there may appear a nystagmic pattern in which the components are reversed in direction and the amplitude of the reaction is reduced. I think that it is entirely possible that this may be an example in the realm of the labyrinth of Dr. Köhler's figural after-effects. I mention it here, because it would be extremely useful if we could explore these figural after-effects in infrahuman forms.

DR. KÖHLER: Since Dr. Halstead has just referred to figural after-effects, and to possible further applications of this concept, I have a good excuse for also returning to this topic.

Surely, once it has been demonstrated that prolonged stimulation causes satiation not only in the visual cortex but also in the post-central area, it seems a plausible assumption that similar effects can occur in virtually all parts of the brain. There are many facts which invite the theorist to give them an interpretation in terms of satiation. It may well be that the observations mentioned by Dr. Halstead belong in this class. Naturally, it remains to be shown that, under particular circumstances, satiation can lead to rhythmic phenomena and also to the reversals which Dr. Halstead has just described.

If satiation accompanies the processes in many parts of the nervous system, the fact deserves attention also for methodological reasons. In psychology, it is customary to present given situations repeatedly, either, as in many cases of learning, because otherwise the level of achievement would be too low, or because we wish to obtain quantitative data which are statistically reliable. Now, repetition makes for strong satiation, and satiation has an obstructing and a distorting influence on events in the satiated area. Hence it is possible that techniques in which certain performances are often repeated actually introduce a factor which distorts and impedes the phenomena to be investigated. I can see no reason why satiation should be absent from memory experiments in which long series of items of a given kind are read a great many times. We know that under these conditions so-called inhibitions are established, and we also know that it is wise to space the repetitions in time, if higher degrees of the obstruction are to be prevented. Obviously, during longer intervals between repetitions satiation would to a degree disappear. From the same point of view, we can readily understand reminiscence, that is, the fact that recall tends to be better when the test is somewhat delayed. I need hardly add that the "position factor" known to all experimenters in this field nicely fits into such a picture. Again, items which do not belong to the same class as the majority clearly suffer less obstruction than does this mass. All inhibitions of this type have proved to be particularly strong when the materials given in a sequence are similar. Satiation, one expects, would be greatly favored by the repetition of similar processes.

It is not quite easy actually to prove that such inhibitions are partly caused by satiation, because in the field of memory we study the statistical behavior of many items, while in perception we investigate satiation in quite specific situations. I will therefore return to the field of perception, and try to show that, when the satiating effects of repetition are not recognized, certain experimental facts may be completely misunderstood. It is a special example which I will now discuss, but the general lesson which it contains will be obvious to any psychologist.

In the early years of this century, Judd in America and Lewis in England proved that the Müller-Lyer illusion is gradually destroyed if its size is measured in many trials. At the time, psychologists still believed that, far from being real visual distortions, such illusions are merely matters of mistaken judgment. Thus it seemed natural to assume that their destruction in repeated trials was caused by better forms of judging, which developed during the repetitions. In other words, it was believed that learning was responsible for the change. This again appeared to confirm the thesis that such illusions are brought about only by erroneous judgments. Actual vision, it was then thought, would not be affected by learning. Now, Judd and Lewis could not know that, quite apart from learning, another factor was also operating in their experiments, namely, satiation. Nowadays, we have to think of this process whenever repeated trials change a given observation. It happens that, in the case of the Müller-Lyer pattern, satiation must gradually reduce the size of the illusion. For within the three angles of the colinear version of the figure, satiation is much stronger than it is in the regions toward which the angles point. As a consequence, the two angles on the side which looks shorter must move away from each other, and the third angle must move toward the middle of the pattern. Thus the difference in the appearance of the left and the right horizontal distances, that is, the illusion, must decrease as the subject inspects the figure in one trial after another.

We have recently subjected this explanation to several tests. First, in most earlier experiments the subjects were allowed to move their eyes while they compared the two distances. Under these circumstances, the particular distribution of satiation to which I have just referred will be established only as a statistical average. If it is actually satiation which destroys the illusion, the process ought to be accelerated under conditions of fixation. This we found to be the case. In Judd's experiments, the illusion did not disappear entirely until his subjects had made 600 to about 1000 measurements. With fixation, the same result was obtained when, on the average, our subjects had made 100 measurements. And yet, the pattern used in our experiments gave a greater illusion than Judd's figure. I must, however, add a qualifying remark and a second observation. If in such tests the illusion is destroyed by satiation, then this factor operates independently of the illusion as such. Thus there is no reason why in all subjects satiation should be strong enough to overcome the illusion completely. The intensity of satiation varies considerably from one subject to another. Actually, we found that, with some subjects, the illusion first rapidly decreased, but that, when this process had continued for a while, the illusion was not further reduced by additional trials. While this fact can readily be understood if satiation is the responsible factor, an interpretation in terms of learning seems much less plausible.

A third observation which points in the same direction follows from the same principle. If, in some instances, satiation may be too weak to overcome the illusion entirely, in others it may be so strong that its final effects go far beyond mere destruction of the illusion. With some subjects, we therefore continued experimentation when the illusion had disappeared. The result was that these subjects soon developed considerable illusions in the opposite direction. It would be hard to explain this fact on the assumption that repetition of trials leads to more adequate ways of comparing the two distances. Fourth, one can replace most measurements of the illusion by periods during which the two parts of the pattern are shown in alternation for mere fixation. We found it possible to destroy the illusion also under these circumstances, although the number of actual measurements was now reduced to a minimum. Fifth, when the illusion has been destroyed it tends to reappear as soon as subjects change their position or the direction of their eyes with regard to the pattern. This fact, which was already known to Judd, is virtually incompatible with the theory that the illusion disappears under the influence of learning. I am not acquainted with any instance of learning in relation to visual patterns in which the result of the process is localized in this fashion.

Sixth, it can easily be demonstrated that, after the destruction of the illusion, there is a localized pattern of satiation in the field, by which the size of the illusion *must* be reduced. When the illusion had disappeared, we replaced the three angles by three short vertical lines which passed through the apices of the angles. Our subjects saw this objectively symmetrical pattern as strikingly asymmetrical. The distance corresponding to the originally shorter-looking part of the illusion pattern was larger, obviously because on this side the limiting lines were displaced away from each other, while the third line was displaced inward. A seventh argument for my interpretation follows from experiments made by Lewis. In some of his experiments, he showed the illusion pattern for only one-fiftieth of a second, the method of constant stimuli being used in the measurements. Although under these conditions his subjects soon found judging easier, the illusion was now hardly affected by continuous experimentation. It seems plausible enough to assume that, with exposures which are so short, no persistent satiation can be established by summation.

This seems to me fairly convincing evidence. Let me repeat. If this happens in the case of the Müller-Lyer illusion, what right have we to assume that satiation does not operate when, for otherwise admirable reasons, we give large numbers of experiments in other cases? In such instances, are we allowed to interpret our findings in terms of what we consider to be our experimental variables alone? The belief that the Müller-Lyer illusion is destroyed by learning has already led to strange conclusions about learning in general—not to mention the fact that it has served to support a mistaken theory of the illusion as such. We may discover that similar mistakes were made when trials were often repeated in other situations.

The Symposium from the Viewpoint of a Clinician

HENRY W. BROSIN*
Department of Psychiatry, The University of Pittsburgh
Western Psychiatric Institute and Clinic

As you know, I did not come prepared to give a formal address but am honored that you have asked me for some extemporaneous comments on the general subject "The Symposium as Seen from the Viewpoint of a Clinician." This would appear to be a relatively simple assignment, because many topics of clinical interest have already been discussed in the formal lectures and the extensive interchange which followed them. However, you will sympathize with me in being discreet because hasty and impromptu impressions about this vast material could hardly do the subject matter justice. This exploratory learning has repeatedly shattered my cerebral organization to the extent that I marvel at the durability of the nervous system to maintain any identity at all!

I am especially grateful to the Hixon Fund Committee for including me, a clinician, although I have little apparent membership in this group of basic scientists whose attitudes toward the world in operation are so different from mine. The members who compose this symposium possess a catholicity, an over-all view, which augurs well for the future of intelligently supported learning. Often parochial attitudes are only too obvious, not only at scientific meetings but in universities and governmental agencies, so that good communication is prevented. For example, psychologists who favor statistical analysis often can find little good in those who utilize projective techniques, even though it would seem that both groups should find ways to bridge the gap separating them. The return at this time—the output as compared to the intake—may be quite small, but we can hope that all skills will make appropriate contributions.

In his summary on Wednesday, Dr. Gerard furnished us with a neat diagram. He differentiated four levels of operation: molecular, neuronal network, cortical-psychological, and social man. The latter levels now require more urgent attention. They must be understood not only as outgrowths of their functions at the cellular and neuronal network levels but at their own distinctive levels with particularly adapted methods and vocabularies. Although the workers in these fields have not yet perfected their means of observing, recording, and comparing their data, Dr. von Neumann's brilliant exposition of man's essentially intuitive relation to the machine provides us with some consolation since this is the familiar position of the clinician. We can profit by his example in not regarding the problem as insurmountable. The four-level diagram also illustrates picturesquely the possibility of influencing levels of widely separated behavior with a minimum of participation by the intermediate ones. This is now done by various shock and drug therapies and may have much wider application, including activities in the reverse direction which are now so popular a focus of interest by internists and psychiatrists. Dr. Klüver hinted at this when he described the possibility of side effects of molecular or neuronal organizations which may cause, in a manner of speaking, extra-cerebral collateral poisonings. Dr. Köhler and Dr. Lashley have, however, documented the wide chasms between these levels of operation.

---

* At the time of this conference Dr. Brosin's affiliation was: Division of Psychiatry, The University of Chicago Department of Medicine.

There is, at present, relatively little communication between those working at a particular level and those working at other levels. However, effective function can be facilitated if a worker in any one area becomes acquainted with the types of problems being studied outside of it. The content and the method employed elsewhere may in many respects be alien and, therefore, forbidding, but in the long run acceleration of total integrated knowledge about human behavior depends on this type of effective interdisciplinary activity. Important examples are furnished in this symposium. One of the finest impressions that I have of this meeting is the acknowledgment made by Dr. Gerard to psychiatrists like Berger and the psychologists who have helped him as a neurophysiologist. Dr. Lorente de Nó's tribute to Dr. Lashley illustrates effective cooperation as distinct from a mere pseudo-aggregate of gentlemen in the same room who have only a social acquaintance but no genuine professional relationship.

Another impressive acquisition is Dr. Lashley's presentation. It will help me directly in answering the questions: *What is the world of a psychologist like? Can you give me some approximation about the tools used?* The student who wonders about the relation of psychology to neurophysiology can be referred to this symposium for ample evidence that these sciences are growing steadily in spite of the pessimism currently voiced by such leaders as Sherrington and Adrian. Boring's conception that the solution to many of these problems will have to wait for a riper time, as reported by Lashley, until the background auras really become much more effective, leads me to recall that the background of the sciences concerned with central nervous system activity has improved markedly in the last twenty-five years. These papers are much more concerned with the problems which have some relation to living and feeling people. They are not dependent on oversimplifications which tend to distort the relevance of an experiment to the point of meaninglessness.

I see no necessary philosophical differences in many of the areas in which the discussants are working. I do not want to indicate that there aren't any; there are, but they can be sharpened into propositions which are meaningful for experiment. I strongly endorse Dr. Lashley's comment about the need for experiment and the superiority of experimentation over abstract argument. I came into the world of psychological ideas in the early twenties. I recall the fanatical intolerance of some groups. Thirty years ago, behaviorists did not write of so extraordinarily complex a world. The early simple Watsonian diagram is incompatible with this new world view. I would also offer that this symposium has achieved remarkably well some correspondence of ideas. At least we are often talking to the same point. However, this isn't enough. We must learn a common vocabulary by living and working together. Reading and experimentation in isolation are inadequate. It may take a century for the men working at molecular and neuronal levels to become thoroughly and intimately acquainted with the world view of a person whose days and nights are steeped in the concepts of man as a social unity.

The plainly tolerant attitude that has characterized this symposium merits more than passing comment. True communication in the sphere of ideas is probably always difficult. It is of great significance that we can meet together without arousing anxiety and discomfort that propel towards flight and refuge. I suggest that one of the most valuable consequences of this meeting may be allied to the manner in which we have carried on our interpersonal relations—however fragile and delicate our intellectual contacts may have been. The achievements in our personal dealings with one another may be more important for this century than the specific contents we have discussed. This is true not only because artificial, economic, and political barriers make communication difficult, but the strain of carrying conflicting loads of interdisciplinary problems militates against one mind's encompassing larger meaningful configurations. We should make it easier for one man to master several related fields by reducing the obstacles as much as possible. It is probable that the most significant innovations will come from one man rather than from a team.

I think that the logic of determinism and the pragmatic study of relations and operations are adequate scientific working methods for the skeptical clinician who deals necessarily with poorly defined complex conditions, even though he himself has not been able to create a useful vocabulary for his needs. There need be little doubt that psychiatry and psychoanalysis are intimately related to both biology and sociology. As investigative techniques in both areas improve, the practice of clinical medicine will faithfully reflect the advances. Most of us hope that the biological roots of man's behavior will not be neglected because of the apparently easier methods offered by sociologists. Even though impressive demonstrations of the importance of environment in determining behavior continue to appear, we do not believe that the body can be explained away in terms of a neutral plastic material. The newer "therapies" employing physical agents open up new fields for studies. Even the distressing practical failures of the various electroshock and surgical methods need not deter us from hoping for better studies about cortical functions which will advance the potentialities for experimental and therapeutic control.

This summary stresses the inner strength of the group. It seems to me that there is much more willingness here than elsewhere to tolerate partial answers. You do not appear to have the acute or permanent need for concrete, sharply defined answers at the cost of reliance upon oversimplified and, therefore, often futile systems. Though not uniform, this greater individual and collective ability to tolerate uncertainty seems to me a healthier attitude. While it may not be applicable to all areas—for I hesitate to paint the picture of absolute harmony—I think that it presages the possibility of a more integrated biological orientation that could include social factors with less distortion of facts. The neurophysiological and psychological concepts presented during the last week are closer to clinical work than the abstract formulations of 1920.

Physicists have worked out a satisfactory method for tolerating apparently irreconcilable views and sharply conflicting data that permits a more comprehensive discussion than is possible by mere face-to-face juxtaposition. "The principle of complementarity" of Niels Bohr encourages examination of conflicting views with the understanding that both may be accurate expositions from a specified point of view, and that the complete rejection of one or the other is not required in order to derive working conclusions.

In spite of the encouraging progress exemplified in these papers, there are some notable gaps. We still do not have much material about those operations labeled "motivation." For example: *Why do people do things? What does the organism do in order to maintain harmony between the internal world and the external world?* We have heard little about emotions and purposes and the relations between them. We have heard less about the basic needs or drives and the nature of these internal-external adjustments of man under stress. In a few places, examples from general psychopathology were cited. There was, however, no treatment of the "anatomy and physiology" of hallucinations, delusions, phobias, obsessions, compulsions, disorders of character and psychosomatic reactions such as anorexia, obesity, hypertension, colitis, and peptic ulcer. We did not hear about research that would help us understand the behavior of a man who is relieved of a peptic ulcer by vagotomy and who then becomes alcoholic or suicidal.

At future meetings we shall probably be able to discuss more meaningfully such social patterns as greed, race hatred, gambling, ambition in bureaucrats, the rearing of children, and similar problems of relational determinism at the social level. It is not beyond hope to anticipate the development of our scientific skills and ethics so that a man who, by virtue of unsatisfactory interpersonal relationships with his employer, develops symptoms on either the somatic, psychological, or social level can be treated appropriately. Currently the solution for such a problem is, all too often, sought by pharmacological or surgical interruption of afferent nervous pathways. We may even, so to speak, refrain from giving atropine to dry the tears of a bereaved parent. I need hardly caution this group that in a complex analysis of personality functions within the field of human relations, social problems per se can and should be handled at that level in our civilization. Decapitation and amputation techniques may appear to be expedient, but they will not be entirely popular as the long-term effects become known. Dr. Klüver's report of the occurrence of endometriosis in a temporal lobectomized monkey is a good example of the dread possibilities. We don't know what kind of a generation we are creating with our techniques of violence in this century. Why does one patient following an operation for brain tumor with good physiological and mechanical results die in a year or two when most similar patients do well? Well-informed surgeons do not hesitate to look into factors of social import to help them find an answer. More psychologists will also when they are familiar with the fact that a human being must maintain viability in the social matrix or suffer distortion and physical death. That everyone must maintain psychological and emotional stabilities in appropriate patterns as well as physical homeostasis is a truism which has not found much popularity in the laboratory.

I introduce now a series of problems which require interdisciplinary cooperation. For lack of simpler methods to study controlled behavior, we place the human organism under stress or, to quote G. Stanley Hall and Adolf Meyer, create the "experiment in nature." My first example is the Rorschach experiment, one of the projective techniques. We ask: Why does response to color reflect the subject's potential for expressing emotion? This is a splendid opportunity for clinicians, psychologists, and neurophysiologists to work together for mutual benefit. Much more sophisticated and, I think, the greatest single act of insight on the part of Rorschach was his description of figures seen in this partially structured material of the ink blot as human movement. Why should these "movement responses" represent the capacity for inward living? Why should the accurately perceived form, the F plus, be probably the best index to ego control? In the twenty-seven years since Rorschach published his monograph, no new theoretical constructs or substantial technical improvement has been devised. For years Dr. Köhler and his colleagues have given us a vocabulary to discuss the properties of configurations. It is regrettable that we have not yet found talent for this purpose in the study of the Rorschach method. It is obvious that we must train experimentalists in this field. The future of good clinical investigation depends upon attracting the people who can appreciate the importance of this type of problem and be willing to live with it.

Mention of these problems calls to mind the many difficulties that are encountered in the selection of research workers, medical students, executives, and others. A few days ago Dr. McCulloch mentioned the man who differs from other men because he has an unusual attention span of four hours. I am not sure that I understand what that means. Olga Rorschach, in writing of her husband, tells of his inability to work at any single task for more than an hour at a time.* He worked a great deal, but hour by hour he changed tasks. In this way he maintained psychological productivity at a very high rate. Without attempting to answer these questions, we might consider the problem of motivation as it pertains to Rorschach. What distinguishes this drive for achievement which, Olga Rorschach said, is the hallmark of a European, and which prompted them to leave Russia after living there for a year, although they loved it very much? She said that he was half consciously and half unconsciously afraid of disturbing his capacity for achievement. He thought that his interest and ability might diminish if he continued to live in a beautiful Russian community. Olga was a Russian; Dr. Klüver prompts me to remark that she spoke like an Oriental.

---

* O. Rorschach. Über das Leben und die Wesensart von Hermann Rorschach. *Schweiz. Arch. Neurol. Psychiat.*, 1944, 53, 1–11.

These remarks are designed to point up the fact that we do not possess an adequate vocabulary or method by which we can describe the series of operations which characterize the ability of the principal speakers of this symposium to maintain their fidelity to a research ideal. If we could accomplish this, we would accelerate the resolution of some of the problems of selection. In spite of the fact that we know a great deal clinically and psychometrically we cannot determine a priori the qualities that would answer the following question: What do these men have in common which enables them, in spite of an infinite number of distractions, to follow faithfully these highly abstract concepts? The men themselves, in their relationship to the material, are much more wonderful than the material. This, I believe, is one of the important questions to the symposium.

But to return now to a problem that may be of especial interest to the neurophysiologist. Rorschach, in his characteristically carefree manner, observed that patients whose psychotic reactions consisted predominantly of kinesthetic hallucinations and imagery responded to his ink blots differently from those without hallucinatory experiences. He was also able to distinguish by their responses between patients with visual hallucinations and those who reacted through auditory routes. The pre-eminent use of kinesthetic and visual functions was correlated with great capacity for introversiveness, for inward living. Let us, for example, imagine a linear scale which represents high introversiveness at one end and little of this potentiality at the other. We can then grade people with respect to this quality and compare their test performances with behavioral patterns; occupational assets; growth and decline of some potential talents; patterns of defense under stress (a psychiatric diagnosis); and alterations with age, mood, fatigue, and other factors which tend to upset homeostasis. Should the observation revolve about a schizophrenic reaction, the quality and type of hallucination would also come under scrutiny. There is evidence to support the soundness of this methodology. It may very well provide the experimental psychologist and the neurophysiologist with a link between central nervous system organization and behavior. These studies would rest upon the reasonable assumption that there are basic and distinctive differences in functions of the central nervous system at the physiological level between a highly introversive person whose distinctive characteristics are identifiable, both in health and disease, and a markedly extratensive person with equally distinctive patterns. The former has the capacity for creating kinesthetic and visual hallucinations while the latter has not. Aging causes a decline in introversive powers as well as in the capacity for hallucinatory experiences. The obvious extratensive is limited to auditory hallucinations. The recently developed techniques in electronics may very well provide data to reveal relevance between these fields when they are implemented with skill and imagination. Dr. Köhler's direct-current electroencephalographic experiments serve as a prototype for this kind of exploration. Another simple example from Rorschach may further illustrate the possibilities. Even though strongly introversive persons produce so-called "movement" responses, they are quite likely to be clumsy and awkward in their motility. On the other hand, extratensive persons with little capacity for producing "movement" responses are more likely to be graceful.

The deterioration of cortical functions associated with degenerative changes or toxic factors decreases the introversive potentialities. In normal aging this process continues slowly and gently so that little loss of function is apparent, under ordinary circumstances, after the fourth decade. In his comparison between the imagery of Goethe and Schiller, Rorschach provides an illustration of the aging process. (Hermann Rorschach, *Psychodiagnostics*, Hans Huber, Berne, Switzerland, 1942, p. 106.) His explanation is, of course, only one of the several tenable hypotheses.

> The loss of capacity for introversion begins in men before the fortieth
> year, and the experience type gradually approaches the extratensive
> thereafter. The perceptive and imagery types should show similar changes.
> A quotation from Stern (p. 373) bears on this point.
> "The visual qualities of Schiller's lyrics were compared with the same
> qualities in Goethe's lyrics by Karl and Marie Groos, using Frank's com-
> putations on Goethe's works. Certain similarities and differences were
> found in the comparisons of the works of the two men. It was found
> that, in his earlier works, Schiller used more than twice as many expres-
> sions referring to visual qualities (computed as rate in 10,000 words of
> text) as did Goethe. In Goethe's works, the number of allusions to visual
> qualities increases with the age of the writer, while in Schiller the number
> decreases considerably; this is especially apparent in regard to expres-
> sions pertaining to brilliance, glare, glitter. Similarities between the two
> authors appear. In both, the ratio between the number of expressions con-
> nected with vivid color and other optical qualities is 1:3, both in the early
> and the later works. As both authors grew older references to red de-
> creased in frequency and expressions involving green increased; in both
> blue was poorly represented, and both used yellow hardly at all. In both

there is an increase of expressions involving lighter shades as age increases."

In another section of his book, Stern quotes the same authors to the effect that Schiller was more strongly acoustic (in imagery) than was Goethe and was far more so than Shakespeare.

"The differences between the two authors and the trends shown in their use of optical imagery may be explained by assuming that Goethe's experience type was originally far more dilated than was Schiller's, that is, it originally spread far more into the introversive sphere than did Schiller's. As both men grew older, kinaesthesias decreased with the result that Goethe then fell into the area of visual types, while Schiller more nearly approached the auditory imagery type. This explains the decrease in expressions of optical qualities in Schiller and the increase of them in Goethe."

A second area for interdisciplinary investigation is in the field of psychosomatic research. From the best evidence available we note that the most significant motivational constellations are deducible from ordinary observable behavior. For example, clinicians are generally familiar with the typical phasic exacerbations and remissions that occur in patients with peptic ulcer, with or without treatment. These patients respond favorably to many and varied types of therapy. In most cases, the efficacy of psychotherapy is probably best understood in terms of the conflict between repressed unconscious passive dependent wishes and the patient's ideal of himself as an independent, self-sufficient adult. Often the psychological defenses take the form of the denial of the desire to be taken care of, the acceptance of many responsibilities, the vigorous striving towards ambitious goals so that autonomy may be attained. These unconscious activities remind us of the "third type of memory" discussed earlier this week. They are extremely useful clinically because they enable us to understand causal relations in a chain of complex events which otherwise would not be explainable by a unified hypothesis. Numerous studies combining psychological and physiological techniques attest to the fruitfulness of these investigations. It is highly likely that a number of syndromes in medicine will be conceptualized, in the near future, as basic psychological problems with concomitant physiological disorders. The well-known observations of Harvey Cushing (1932), who showed the direct effect of various types of brain injury on gastric tissue, elicit special interest from the neurophysiologist. More recent studies on lobotomized patients may help us further in understanding these relations.

A third group of problems worthy of interdisciplinary study is centered around the subjective state of the observer in the experiment. Astronomers and physicists were the first to direct serious attention to the errors introduced into an experiment by the all-too-human limitations of a single man. The safeguards that they were able to provide have served them well. However, we have not yet been able to perfect our corrective devices in the study of behavior. The problem is particularly acute in the therapeutic situation. Here, one complex organism, the physician, is in relation to another complex organism, the patient. Now, more than at any time in the past, the attitudes, both positive and negative, of the doctor toward the patient are under careful scrutiny. Clinical psychologists, as well as psychiatrists, are becoming more aware that the experimentalist's attitudes are proper objects for study in order to understand the development of the relationship between the two principal figures in a therapeutic "experiment." The mechanics of the psychiatric interview are also being studied much more systematically with the aid of recording devices. We may expect progress to be made in the most basic of our clinical tools —the interview—so that we may operate with greater certitude.

Patients with organic disorders of the cerebrum provide another rich experimental field. In this situation man may be regarded as an experimental animal and is available, for the first time under somewhat controlled conditions, for wide exploration. While I deplore the unreasonable recourse to lobotomy, I wish we could study patients who have undergone this mutilating operation by the methods presented here this week. The failure to do so thus far is our real tragedy. Most patients are operated on with inadequate pre- or postoperative studies by a trained psychologist. Furthermore, as far as I know, there are probably less than fifty careful reports by psychoanalysts on patients with cerebral deficit. It is simple to understand why there are so few. No one wants to make a big investment of his time and interest in a patient in whom the potentialities for good therapeutic results seem small. As a remedy for this scarcity of necessary information, I think that we must remunerate full-time research psychiatrists and psychoanalysts sufficiently so that these men will be willing to work here. I would heartily endorse any program in which truly research-minded, well-trained psychoanalysts would study a number of these patients.

Dr. Lashley said that temporal integration is the most important and most neglected field in neurophysiology. It seems to me that this is equally true in psychopathology. If we understood the integration of personality functioning on a time axis, prediction and control would be markedly improved. We have much evidence now that clinicians are able to predict successfully the progress of a patient from relatively small samples of behavior and that, occasionally, much can be done to accelerate therapy.

In spite of the progress made in describing some properties of ego functions, there exist many problems at the level of social interaction which need elucidation. Presumably, after we identify ego functions at various levels of activity, there will continue to be the problem of intricate interpersonal relations which will require some type of understanding and control. Current clinical bias favors the view that the basic and permanent behavioral patterns, insofar as they are not genetically or prenatally determined, are laid down in the first four to six years of life. The chemical embryological studies suggested by Doctors Lashley and Thorndike can do much to elucidate the characteristics of the immature brain. Learning and memory during the first two years of life may be qualitatively different from the same functions which we are studying at a later time. If this be true, then we must focus on methods permitting us information from this area in order to find the causes of psychoses and neuroses at the adult level. After fifty years of investigation we know that we must get back more and more to the earliest years of the patient's life. The potentiality for schizophrenia, for instance, seems to be established at that time.

In this connection, I was impressed by the discussions of the hierarchical patterns in the central nervous system. These discussions included the description of the lower patterns with relatively independent structure and their primary rhythmic activity which may be modified and distorted from above. All studies that can elucidate the nature of our more primitive central nervous system organizations and their controls will do much to bridge the gap between psychiatry and physiology. It must be remembered that some of these functions, including the endocrine, are designated as operating units by psychiatrists. In this stage of our development, our vocabulary is necessarily crude. In order to talk about this order of events, the biological reservoir, we say "an instinct," "a drive," "an urge," or even "it." Freud (not to mention Bleuler, Schilder, and H. S. Sullivan) received endless criticism from the sociologically minded people who felt that he had disagreeably revealed his essentially organic-neurologic bias by insisting upon the biological nature of the Id. In speaking of the activity in this area, Freud described something very curious. Emotionally charged ideas here maintain distinctive identity as well as the ability to influence behavior and yet, short of complete exposure, remain essentially unaltered by time and activity. Here is an illogical world, free from the space-time relations of ordinary geometry, which presents another problem to the neurophysiologist in analyzing these phenomena. The chemical embryologist, anatomist, and endocrinologist may eventually help the neurophysiologist to give real meaning to this concept of constitution.

In 1929, in his *A History of Experimental Psychology*, Boring said that the application of the experimental method to human mentality is "the great outstanding event in the history of the study of mind." He stated that psychologists were now relieved of the necessity of defending themselves against adverse criticism. They might, he asserted, agree with some of their critics that progress has been slow and disappointing, but they had the huge burden of philosophy to drag along, both administratively and idealogically. One formulation might be: the more philosophy, the more abstractions; the less real work, the less insight. However, aside from these factors, what are the basic handicaps? Boring offers two brave guesses: lack of conceptual unity and lack of a great man.

Some of you may feel that, in the week that has gone, you have seen outlined the background which may provide the long-sought-for conceptual unity. Boring warned that mere industry is not enough. Patient application and the pyramiding of unknowns do not add to our knowledge. Insight and specific methods are needed. As regards Boring's second requirement, that psychology has never had to itself a truly great man, I would be bold enough to say, for psychiatry, that we have our great man—Freud. He has provided us with ample material and techniques. The exploitation of the free-association technique and the inquiry into the meaning of psychodynamic social patterns can keep us occupied productively for many years. I wonder whether psychology will not find its great man in the person who can utilize the Freudian concepts and bring them into closer approximation with the ideals of Wundt.

Name Index

(The names of the speakers at the symposium are set in capital and small capital letters. Italicized page numbers are the pages on which there are bibliographical items.)

Ackerly, S., 266
Adrian, E. D., 94, 95, 141, 209, *229*, 235, 281, 290
Akelaitis, A. J., 132, *135*
Alcmaeon, 43
Allen, W. F., *178*
Allers, W. D., 137
Alles, G. A., 197, 198
Asch, S. E., 243

Bailey, P., 51, 250, *270*
Bard, P., *178*
Barker, S. H., *57*
Barrera, S. E., 166, *179*
Bartelmez, G. W., 269
Bartley, S. H., 95, 96
Beadle, G. W., 269, *270*
Becher, E., 130, *135*
Bell, C., *57*
Bender, M. B., 242
Berger, H., 235, 290
Bernard, C., 193, 194
Berry, C. M., *179*
Bethe, A., 123, *135*, 141
Bigelow, J., *57*
Binet, A., 245, 246, 247, 263, *270*
Bishop, G. H., 95, 100
Bleuler, E., 299
Bohr, N., 292
Bonin, G. von, 50, 51, 250
Boole, G., 106
Boring, E. G., 117, *135*, 144, 300
Breasted, J. H., 244, 245, *270*
Bremer, F., 141
Broca, P., 120
Brodal, A., 164, *179*
Brookhart, J. M., *179*
Brosin, H. W., 63, 69, 72, 77, 79, 82–83, 89, 99, 106, 182, 289–300
Brown, Sanger, 152, 153, *179*
Brown, T. Graham, 85, 126, 127, *135*
Brunswik, E., 261, *270*
Bubnoff, N., 56
Bucy, P. C., 151, 163, *179, 180, 181*
Buddenbrock, W. von, 123, *135*
Bullock, T. H., 81
Bush, V., 5

Cajal, R. y S., 51, 55, *57*, 60
Cannon, W. B., 48
Caspersson, T., *270*
Cattell, J. McK., 281
Chaikoff, I. L., 164, *179*
Chamberlain, A. F., 112, *135*
Clark, A. J., 178, *179*, 198
Clark, W. E. Le Gros, 131, *135*
Coghill, G. E., 141
Conel, J. L., 247, *271*, 278, 279
Craik, G. L., 40, *40*
Crosby, E. C., *271*
Culler, E. A., 60, 138
Cushing, H., 297

Darwin, C., 251
Degkwitz, R., 169, *179*
De Jong, H., 167, *179*
Denny-Brown, D., 179
Dey, F. L., 165, *179*
Dick, F. M., *182*
Dinnerstein, D., *229*
Dusser de Barenne, J. G., 52, 280

Ehrlich, G., 76
Emerson, E., 282
Emery, D., *230*
Epstein, I., 118, *135*

Ferraro, A., 166, *179*

Ferrier, D., 153, *179*
Fishback, J., *230*
Fisher, C., *179*
Flechsig, P., 247, *271*, 274, 278, 279
Flourens, M. J. P., 276
Forbes, A., *181*
Fournie, 113, *135*
Fränkel, S., 169, *179*
Frantz, R. H., *182*
Freud, S., 119, 299, 300
Fritsch, G., *135*
Fröhlich, A., 97

Gasser, H. S., 83
Gastaut, H., 95
Gellhorn, E., *57*, *181*
GERARD, R. W., 33, 34, 63, 64–65, 77–78, 94, 96, 106–109, 138, 194, 235–237, 238
Gerard, R. W., 211, *229*, *230*, *237*
Gibson, J. J., 204, 205, 206, *229*
Goddard, H. H., 245
Goethe, J. W. von, 243, 296, 297
Goldstein, K., 133, *135*, 254, *271*
Grinstein, M., *179*
Gross, Karl, 296
Gross, Marie, 296
Grünbaum, A. A., 197
Gunn, J. A., 168, 169, *179*

Hall, G. S., 294
HALSTEAD, W. C., 40–41, 88, 95, 96, 99, 138–139, 244–272, 273, 274, 275–276, 285
Halstead, W. C., 254, 255, 257, 261, 264, 265, 266, *271*
Harlow, H. F., 259, *271*
Hartelius, H., 270, *271*
Hartung, W. H., *180*
Hawkinson, V., *182*
Hebb, D. O., 246, 252, *271*
Heidenhain, R., 56
Held, R., 213, 214, *230*
Hinsberg, K., 180
Hiscoe, B., 281, *281*
Hitzig, E., *135*, 276
Holst, N. v., 126, 127, *135*, 141
Huber, G. C., *271*
Hydén, A., 270, *271*, 282

Ingebritzen, O. C., 125, *135*

Jacobsen, E., 120, *135*
Jakob, C., 247, *271*
Jarcho, L. W., *181*
Jasper, H. H., 88, 228, *229*
Jeans, J., 43
JEFFRESS, L. A., 85
Johnson, H. C., 80
Judd, D. B., 286, 287

Kappers, C. U. A., *271*
Katz, J., 269
Kennard, M. A., 272
Kleist, K., 164, *180*
KLÜVER, H., 78, 84–87, 88, 96, 97, 136–137, 147–182, 183, 196–199, 242–243, 274–275
Klüver, H., 147, 149, 151, 154, 163, 164, 165, 167, 169, 170, 171, 172, 177, 178, *179*, *180*, *181*, 265
KÖHLER, W., 65–69, 93, 94, 95–96, 137–138, 196, 200–230, 231–233, 234, 237–238, 239–240, 241, 242, 243, 285–288
Köhler, W., 200, 202, 205, 207, 210, 211, 213, 214, *229*, *230*, 251, *271*
Kubo, H., 177, *181*

Larson, P. S., *179*
LASHLEY, K. S., 37, 40, 70–72, 82, 112–136, 144–146, 195–196, 230–231, 272–273, 280–281
Lashley, K. S., 122, *135*, *136*, 206, 230, 251, 253, 265, *271*
Leao, A. A. P., 237
Leimdorfer, A., 194
Leininger, C. R., *179*, *181*
Lettvin, J. V., 57, 59, 60, 61, 64
Lewis, E. O., 286, 288
Libet, B., 211, *229*, 237
LIDDELL, H. S., 83–84, 240–241, 284, 285
LINDSLEY, D. B., 87–89, 143–144, 241–242, 277–279
Lindsley, D. B., 94, 143
Liston, M. D., *230*
Lloyd, D., 55, 60
LORENTE DE NÓ, R., 41, 57–58, 62, 82, 131, 132, 139–140, 233–235, 238
Lorente de Nó, R., 46, 51, 57, 74, 75, 79, 82, 279
Lowenfield, V., 273

McColl, H., *57*
McCULLOCH, W. S., 32–33, 37, 39, 42–57, 58–61, 62, 64, 82, 85, 87, 88, 96, 97, 99–106, 142–143, 193–195, 238–239, 273–274
McCulloch, W. S., 21–24, 33, 34, 41, 45, 52, *57*, 87, 134, *182*, *271*
Magnus, R., 124
Marquis, D., 60
Mathes, M. E., *179*
Matthews, B. H. C., 95, *229*
Max, L. W., 114, 120, *136*
Maxwell, C., *57*
Merten, R., *180*
Meyer, A., *181*, 294
Mill, J. S., 44
Mitchell, S. Weir, 199
Monakow, C. von, 136, 145, 163, *181*, 275
Morison, B. R., *181*
Mountcastle, V. B., *178*
Mourgue, R., *181*
Munk, H., 197, 276
Murphy, J. P., *181*

Newton, I., 43
NIELSEN, J. M., 182–193, 276–277
Nielsen, J. M., 183, *183*, *186*, *189*, *192*, *193*, 255, *271*
Nissl, 90
Noteboom, L., *181*

Obrador, S., *181*

Panizza, B., 276
Papez, J. W., 163, 164, *181*
PAULING, L., 91–94
Pauling, L., 76, 266, 269, *271*, 282
Pavlov, I. P., 60, 83, 240
Penfield, W., *181*
Pick, A., 113, 115, 117, *136*, 145
Pitts, W., 21–24, 32, 33, 34, 41, 45, 47, 55, *57*, 87
Planck, M., 43
Plato, 49
Polyak, S. L., 78, 148
Prosser, C. L., 141

Querido, A., 171, 172, *181*
Quinn, C. E., *230*
Ranson, S. W., 165, *179*, *181*
Reichert, F. L., *179*
Reiter, L., 261, *270*
Renshaw, B., *181*
Reynolds, B., 107
Richter, C. P., 77, 165, *181*
Rodewald, W., *180*, *181*
Roizin, L., 181
Rorschach, H., *294*, 296
Rorschach, O., *294*, 295
Rose, J. E., *181*
Rosenblueth, A., 48, 55, *57*
Russell, B., 142
Russell, J. T., 206, *230*

Samuels, M. R., 261, *271*
Sargeant, W. E., *230*
Schäfer, E. A., 152, 153, 154, *179*, *181*, 276
Schilder, P., 299
Schiller, J. C. F. von, 296, 297
Schwartz, S., *179*, *182*
Sciarra, D., *179*
Scott, G. G., *230*
Sherrington, C. S., 124, 125, 129, *136* 290
Shurriger, P. S., 60
Simon, T., *270*
Smith, E., 244, 245
Smith, J. R., 87, 279
Smith, W. K., *181*, *182*
Spearman, C., 41, 254, 263, 264, *271*
Speransky, A. D., 170, *182*, 274
Sperry, R. W., 74, 89
Spielmeyer, W., 168, 169, *182*
Spinoza, B. de, 54
Spitzer, A., 150, *182*
Spitzer, H. F., 150
Spooner, W. A., 119
Stern, W., 296, 297
Stoll, W. A., 168, *182*
STROUD, J. M., 97–98, 99
Stroud, J. M., *99*
Sullivan, H. S., 299
Szent-Györgyi, A., 269, 270, *271*

Terman, L., 246
Teuber, H. L., 242
Thorndike, E. L., 299
Thorsen, A. M., 114, *136*
Thurstone, L. L., 41, 254, 263, 264, *271*
Titchener, E. B., 115, 117, *136*

Toman, J. E. P., 95, 96
Turing, 26–28, 32
Tyler, D. B., 93

VAN HARREVELD, A., 297–280, 281
Vega, G. F., 39
Verhoeff, F. H., 204, *230*
Vogt, O., 137, 247, 248
VON NEUMANN, JOHN, 1–31, 33, 34, 35–36, 37, 38–39, 41, 58, 61, 62–63, 96, 109–111, 132, 232

Wade, M., *271*
Waldenström, J., *182*
Walker, A. E., *182*
Wallach, H., 202, 204, 205, 207, 210, 211, *230*
Walters, W., 95
Wang, G. H., 95
Ward, A., 60
Ward, A. A., Jr., *182*
Washburn, M. F., 114, *136*
Watson, C. J., 174, *179*, *182*
Watson, J. B., 114, 120, *136*, 249, *271*, 275, 278
Weidenreich, F., 275
Weil, A., 274
WEISS, P., 34, 36, 72–77, 78–79, 89–91, 94, 140–141, 196, 240, 281–282
Weiss, P., 281, *281*
Wernicke, C., 120, 151
Wertham, F., 166, *182*
Wiener, N., 43, 44, 48, 55, *57*, 60, 142 255, *271*
WIERSMA, C. A. G., 79–82, 284
Witkin, H. A., 243
Wittgenstein, L., *57*
WOODBURY, L., 89, 282–284
Woolsey, C. N., *181*, *182*, *272*
Wundt, W., 300

Yerkes, R. M., 244, 253, *272*

Subject Index

A factor, *see* Intelligence
Abbott Memorial Fund, 147
Abstraction, 40
Acetylcholine, 167
Acoustic nucleus, 234
Adrenergic neurons, 75
After-effects, figural, 200 ff.
  subcortical, 234
After potentials, 222 (*footnote*)
Agnosia, 141, 182, 183, 255
  for animate objects, 187, 188, 190
  for inanimate objects, 185
Agrammatism, 113
Agraphia, 255
All or none principle, 4, 9, 10, 46
Alpha rhythm, 51, 87, 94, 100, 101, 209, 235
  and hysterical blindness, 104
  and myelinization, 278–279
  as scanning, 51
  blocking of, 87–89, 94, 95, 144, 227
  driving of, 95, 255, 268
  in children, 97
  in infants, 278
American Philosophical Society, 213
Amino acids, 92
Amnesia, 64
Amplifier, direct-current, 213
Amputation, effect of, on stepping order, 123–124
Amygdala, 143, 164
Analogy (analogue) devices, 5, 6, 43
  electrical vs. mechanical, 7
Anatomic variability, 70
Anoxia, 256, 280
Ansa lenticularis, 143
Antigens, 92
Aphasia, 113, 120, 121, 145
Aphemia, 120
Appetitive circuits, 49
Apraxia, 255
Area, cortical, *see* Cortex, cerebral, areas of
Arnold's bundle, 163
Asphyxia, spinal, 280
Associationism, 53, 141
Associative chain theories, 113
Atomism, 65
Auditory cortex, *see* Cortex, areas of
Automata, comparison with living systems, 2, 9, 12
  logic of, 1, 17
  possibility of analytical theory of, 16–19
Axial system, 14
Axiomatic method, 2

Baillarger, strip of, 51
Basal ganglia, 143
Behavior, cerebral aspects of, 108
  changes in, following lobectomy, 151 ff.
  embryological development of, 73
  irrational elements in, 107, 263
  molar, molecular, and neuronal aspects of, 108
Behaviorism, 117
Berger rhythm, *see* Alpha rhythm
Bewustseinslage, 117
Binary system, 4
Black box, 2, 6, 11, 22, 26
Blindness, hysterical, 104
Blind spots, *see* Scotomata
Blocking of alpha rhythm, *see* Alpha rhythm
Blood sugar, stimulus equivalence for, 194
Blueprints, 109, 110
Boltzmann's thermodynamics, 17
Boolian algebra, 106
Bowing, violin, 127
Brain, early history of the concept, 245
Brain potentials, magnitude of, 237
Brightness and flicker frequency, 98
Broca's aphemia, 120

Subject Index

Bulbocapnine, 166, 167
Bush differential analyzer, 6

C factor, *see* Intelligence
Catalepsy, 166, 167
Catatonia, experimental, 166
Catatonizing nucleus, 167
Caudate nucleus, 142
Centering reflex, 86
Cephalic ganglion, 244
Cerebellum, projection of, 142
Cerebral cortex, *see* Cortex, cerebral
Cerebral lesions, effects of, 71
Cerebral peduncles, 142
Chain reflex theory, 117
Change in synapse, 92, 97
Chemical factors in neuronal behavior, 76–77
Chemical specificity, 78
Chess playing, blindfold, 125
Chicago Neurological Society, 152
Chinese language, 113
Cholinergic neurons, 75, 78
Chromosomes, 55
Chronaxie of brain, 283, 284
Chronogenic localization, 136
Cingular gyrus, 164
Cold Spring Harbor Symposia, 100
Color response, 294
Color vision, 149
Commonwealth Fund, 147
Communication theory, 6
Complementarity, 92, 292
Complexity, degree of, 4
Complication, concept of, 25, 31
Computing machines, 3, 4, 5
  precision requirements, 4
  present and future size of, 12
Conditioned reflex, 60, 83–84, 241
Consciousness, 54, 99
  motor theory of, 114
Continuity physics, 211
Continuous quantities, digitalization of, 19–20
Contour vs. field in blocking, 94
Coproporphyrin, 173
Corneal reflex, 89
Corpus callosum, 132, 157
Corruption, definition of, 44
  in communication, 45
  in muscle response, 45
Cortex (cerebellar), 55, 142
Cortex (cerebral), 55, 105, 113, 135
  areas of *6*, 86, 277
    17, 85, 86
    18, 71, 87, 185
    19, 71, 87, 138
    32, 86
    auditory, 50, 70, 163
    Baillarger's strip, 51
    calcarine, 184
    face-motor, 56
    frontal, 264 ff., 276
    occipital, 147, 183, 185, 194, 275
    olfactory, 163
    parietal, 275
    prefrontal, 195–196, 249
    prestriate, 71
    silent, 250
    striate, 51, 52, 71, 131, 132, 148, 195
    suppressor, 70, 71
    temporal, 70, 150 ff.
    visual, 48, 104, 131
      primary, 85
      secondary, 85
  interchangeability of, 104
  sensory projection on, 55
Counting vs. digitalization, 20, 21
Crayfish, nervous system of, 79–82
Cree language, 112, 113
Cybernetics, 255
Cytochemistry, 78

D factor, *see* Intelligence
Degeneration, Marchi, 131
Delay, synaptic, 82
Depth effect in satiation, 207
Descending systems, 105
Determining tendency, 117
Differential analyzer, 6
Diffusion currents, 210
Digital element, analogy aspect of, 10–11
Digital machines, 5, 7, 43
  reduction of noise level in, 9
Digitalization of continuous quantities, 19–20
Diphasic brain potentials, 238
Direct-current brain potentials, 211, 237, 239
Displacements, 118

Driving of alpha rhythm, *see* Alpha rhythm
Drugs, selective action of, 75, 166 ff.
Dualism, 69

Earthworm, learning in, 244, 253
EEG, 95, 241
Ekklisis, 163
Electrodes, 213
Electroencephalogram, 95, 143, 241
  see also Alpha rhythm
Electrotonus and satiation, 212
Elements, computor vs. nervous system, 13, 14
  mixed nature of, 10
  number of, in body, 3
    in computors, 3
  of nervous system axiomatized, 2
Embryonic development of behavior, 73
Encyclopaedia Britannica, 55
Endometriosis in monkey, 165
ENIAC, 12, 14
Entropy, relation to information, 43
Envelopes vs. slow waves, 235–236
Enzymes, 92
Epilepsy, 95
Epinephrine, 167
Equipotentiality, 145–146
Equivalence of stimuli, 169, 170, 196, 274
Error, correction of, 34, 35, 36, 37
  cumulative, 38, 39
  foreseen and unforeseen, 37
  treatment of in digital computors, 18, 19
    in living organisms, 18, 19
  vs. certainty, 46
  vs. statistical precision, 37
Error-operated devices, 48
Errors in typing, etc., 118
Extinction, 104
Extracerebral mechanisms in behavior, 171

F response, 294
Facilitation, 104
Factor analysis, 254
Feedback, negative or inverse, 47, 48, 56
  positive, 58
Fetus, EEG in, 144
Field physics, 67–69
Field theory in perception, 230
Field vs. impulse interpretation, 65, 102–104
Figural after effects, 200 ff.
  brightness, 208
  curvature, 205
  depth, 207
  direction of lines, 204
  displacement, 201
  distance, 205
  kinesthesis, 206–207
  size, 202
    of circles, 203
Flicker, 95–96
  brightness of, 98
Fluorescence of porphyrin, 172 ff.
Formal logic, 16
Formal neural networks, 21
French language, 113, 118
Frequency of stimulation, movement and, 56
Frog brain, potentials in, 138
Frontal eye fields, 71
Frontal lobes, 257 ff., 272

Gender in language, 118
Genes, 92, 196
Geniculate bodies, 48, 160
Geniculostriate system, 147, 149, 150
German language, 113, 118
Gestalt psychology, 53
Goal, nature of, 67–68
Grain in nervous system, 103
Group theory, 53

Habit complexity, 253
Hallucinations, 195, 196, 197
  auditory, 295
  kinesthetic, 295
  suppressed by succinate, 195, 197
  visual, 295
Hallucinations pédonculaires, 172
Hemianopia, 184
Hemiplegia, 121
Hemoglobin in leguminous plants, 178
Heschl's gyrus, 49
Hippocampus, 150, 163–164
Hixon Fund, 289
Hog Latin, 119

Subject Index

Homeostasis, 48, 150
Hyperalgesia, 280, 281
Hyperthyroidism, 52
Hypophysis, 165
Hypothalamus, 165
Hysterical blindness, 104

I factor, *see* Intelligence
I object, 200
I.B.M. Corporation, 12
Ideas—defined as invariants, 42
Idiotropic nervous system, 150
Imageless thought, 115
Imagery and age, 296–297
Images, 117
Immunochemistry, 92
Indeterminacy in micro events, 91
Inert period, 80
Infantile paralysis, motor coordination in, 74
Inflection in language, 113
Information as negative entropy, 43
Insulin, 164, 165, 194
Integration, mechanism of, 130
Intelligence, activity, 246
  biological, 254
  brain lesions and, 247
  brain structure and, 247
  factors, *A*, 255–256, 264–268
    C, 256–257
    D, 255, 268
    g, 264
    I, 264
    P, 255, 268
  level, 246
  psychometric, 254
Intention, 11
Interaction, spatial and temporal, 128
Internuncials, 105
Intramolecular forces, 93
Invariance, 42
  of musical chords, 49–51
  of shape, 51
Invariants, 53, 101
Isomorphism, 68, 69, 231
  of visual cortex, 232

Kapper's law, 44
Kekawewechetushekamikowanowow, 112
Klisis, 163

Labyrinth, 139
Lateral geniculate bodies, 131, 160, 234
Lobectomy, occipital, 147
  parietal, 275
  temporal, 151–163, 275
  visual area, 131
Lobotomy, 298
Lock and key relation, 92
Logic vs. neurology, 24
Luminous flux, response to, 147–148

Magnitude, orders of, 3
Magyar language, 118
Marchi degeneration, 131
Mass action, 70
Maturation vs. experience, 64
McCulloch-Pitts theory, 21 ff., 33–34, 41, 87
Medial geniculate bodies, 160
Melody, playing backward, 129
Memory, 41, 107
  organ, 64
  reverberatory, 58–63, 101
  storage in, 63
  switching organs as, 41
  three kinds of, 101–102
  visual, after cortical damage, 72
Memory trace, pattern currents in, 231
Mescaline, 167, 168, 195, 197
Mesencephalon, splitting of, 166
Migraine, 97
Molecular shape, 76
Molecular structure, 92–94
Motion, after-sensation of, 125
Motor aphasia, 120
Motor coordination, 74
Motor horn cells, 105
Motor patterns, central control of, 122, 123
Motor theory of thought, 114
Movement responses, 294, 296
Müller-Lyer illusion, 286 ff.
Music, rhythm in, 126–127
Myelinization, and alpha rhythm, 278
  and learning, 249
  and stains, 274

Nerve impulse as atomic proposition, 47
Nervous system, a hierarchical system, 74

Nervous system, digital and analogous features of, 9
  flexibility of, 109
  statistical properties of, 103
Neural networks, 21
Neurology vs. logic, 24
Neurons, chemical differences in, 75
  growth of, 281
  number of, 3, 13
  protoplasm flow in, 281
  structural differences in, 75
Neurosis produced by conditioning, 84
Niagara Falls, 54, 55
Noise in computors, 5, 8, 9
Noise level, 6
Nonsense syllables, 116
Normalization, 243
Notation, efficiency vs. errors, 21
Nucleoproteins, 269
Nystagmus, 139
  as figural after effect, 285
  optokinetic, 54

Occipital lobe, lesions of, 183
Oculomotor nucleus, 48
Off effects, 101, 211
Oikotropic nervous system, 150
On effects, 101, 211
Operculum, 70
Optic nerve, number of cells in, 131
Optic tract, number of cells in, 132
Organisms, analogy and digital aspects of, 9
  compared with computors, 12
Output vs. input, 63

P factor, *see* Intelligence
Pace-setter, 123
Parietal lobes vs. occipital lobes, 275
Pattern vision and blocking, 88–89
Peduncles, role in hallucination, 172
Peripheral chain theory, 117
*p*H, 93
Phonetics of psychotic patients, 136
Pituitary, 165
Pneumotaxic center, 143
Poisoning, collateral or parallel, 172, 177
Porphyria, 174 ff.
Porphyrin, in adrenal gland, 175
  in brain, 172
Porphyrin, in leguminous plants, 177–178
  in nerve, 173
  metabolism of, 172
Posture, control of, 125
Potentials, direct-current, in brain, 211 ff.
Potential theory, 211
Precentral gyrus, 121
Precision, digital vs. analogy machines, 9
  in nervous system, 91
  in neural path, 72–73
  requirements for in computers, 4–5, 37–39
Prediction, 54
Presbyophrenia, 59
Priming of expressive units, 119, 122
Projection, anatomical, 234
  physiological, 234
Projective techniques, 294
Pronunciation, act of, 115
Protein chains, 269–270
Protoplasm flow in neurons, 281
Psychic blindness, 151, 197
Psychic surgery, 293
Psychogalvanic response, 226, 241–242
Psycho-histological parallelism, 166
Psychometric intelligence, 254
Psychophysical parallelism, 166
Psychosomatic reactions, 293
Pulvinar, 158
Putamen, 143, 157

Quantization, 102–104

Reflex, conditioned, 60, 83–84, 241
  gaze centering, 48
  postural, 125
Reflex arc, monosynaptic, 55
Refractory period, of brain, 283
  of neuron, 46
Relational determination, 210 ff.
Relay organ, 11, 12
Relearning, 73–74
Reliability in computers, 4, 18–19, 37–39
Retina, cells of, 131
Retinal changes following lobectomy, 148

Reverberatory circuits, 47, 58, 82, 86, 115, 131
  as memory, 62, 101
Reversibility of motor patterns, 124, 140
Rhythmic action, 126, 141, 146
Rickets, 169
Righting reactions, 125
Rorschach experiment, 294
Round-off error, 8

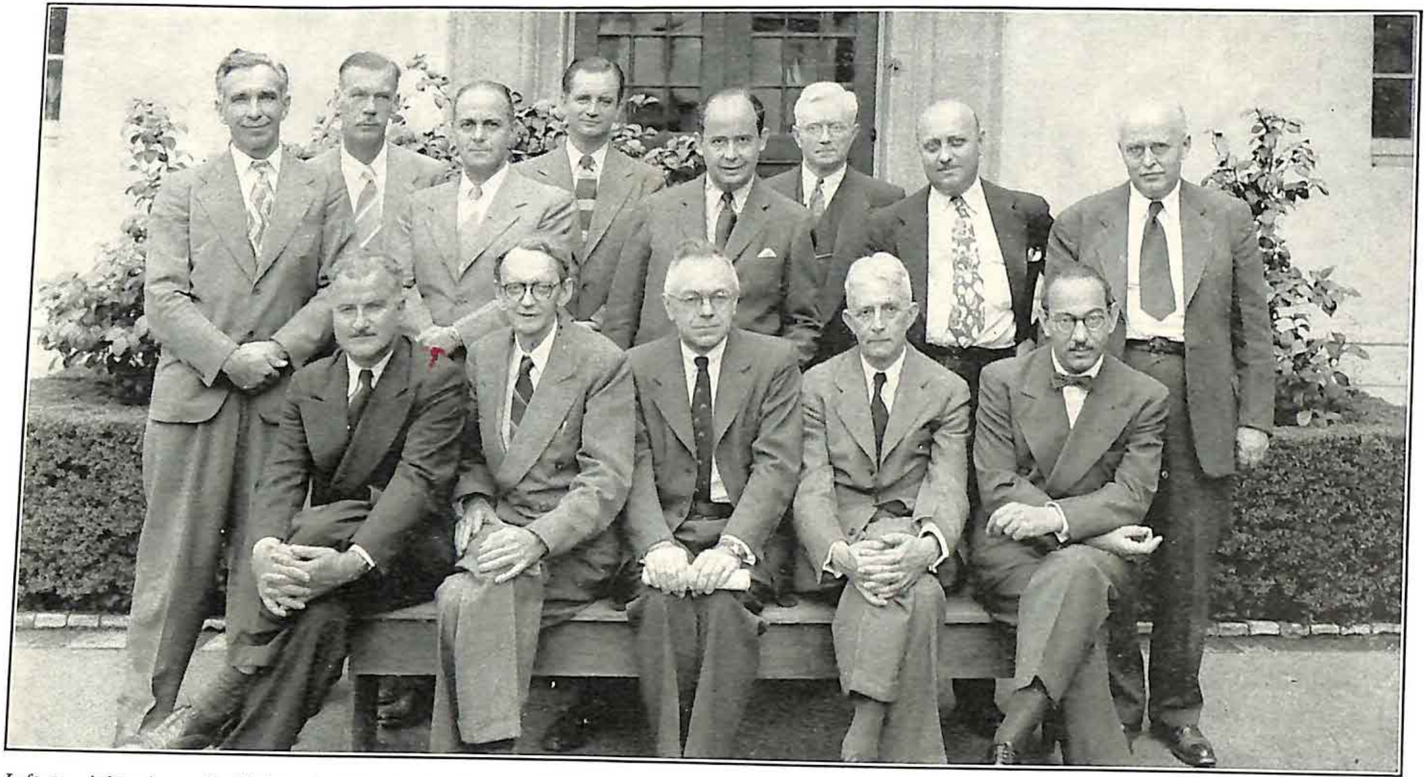
Sampling of information, 45
Satiation, 201 ff., 233
  as tissue polarization, 233
  equivalent to electrotonus, 212
Scanning device, 51, 64, 97–98, 100
Scanning mechanism in speech, 129–130
Scar tissue in cortical lesions, 272
Schizophrenia, 299
Schizophrenic reaction, 295
Scotomata, 52
  scintillating, 96, 97
  visual displacement and, 242
Selectivity, dietary, 77
  of drugs, 75, 166 ff.
Self, 67–68
Self-reproduction, concept of, 25
  genes, 30, 31
  mutation, 30, 31
  of automata, 28–31
Servomechanism, 48
Shape, perception of, 128
Shape of nerve cells, 77, 79
Signal-to-noise ratio, 5–6
Silent areas, 250
Silver, injection of, 171
Sioux language, 113
Size, categories of, 14, 92
Size limits in computers, 15
Slow waves vs. envelopes, 228, 235, 236
Space coordinates, 124–126
Space of implication, 142
Spatial and temporal order, interchangeability of, 128
Spatial summation, 46
Spinal cord, double hemisection of, 125
  learning in, 60–61
Spoonerisms, 119
SSEC, 12
Stellate ganglion, 166
Stepping, order of, 123
Stick insect, 123
Stimulus, frequency of, and movement, 143
Stimulus equivalence, 70, 101, 169, 170
Striate area, *see* Cortex, areas of
Structuralist School, 117
Strychnine, and learning, 280, 281
  spikes, 52, 239
  vs. caffeine, 280
Strychninization of cord, 280
Subliminal stimulation of digital organ, 10
Summation, spatial, 46
Superior colliculus, 48, 85, 195
Superior longitudinal fasciculus, 132
Supernumerary limbs, 89
Suppressor area, *see* Cortex, areas of
Sweep, 100
Switching organ, 11, 12
Sylvius, fissure of, 51
Synapse, delay at, 82
Syntax, general problem of, 121–122

T-object, 200
Tapping, accuracy of, 98
Teeth, trauma to, 107
Teleological mechanisms, 48
Templates in neural cytoplasm, 282
Temporal and spatial order, interchangeability of, 128
Temporal integration, 113
Temporal lobectomy, *see* Lobectomy
Temporal valence, 116
Thalamus, 71, 158
Tonus, postural, 124
Tracts, long fiber, 132
  temporofrontal, 132
  temporo-olfactory, 163
Transformations, 53
Tremor, 143
Tuber cinereum, 170
Turing's computing machine, 26–28, 32, 33
Types of nerve cell, 75, 79

Uncertainty, tolerance of, 292
Uncus, 157, 164
Universal automaton, 26–27
U. S. Army Ordnance Department, 12
Value, 68

Value anomaly, 49
Variability, anatomic, 70
Viking Fund, 213
Visual agnosia, *see* Agnosia
Visual area, *see* Cortex, areas of
Visual discrimination, 131
Visual sector, 150
Vogt's Institute, 137
Volume conductor, 211
Walking-stick insect, 123
Wave analogy for brain action, 133
Wernicke's aphasia, 120
Whip-snapping movements, 123
Wisdom and the frontal lobes, 277
Word nearness, 137
Word order, 113
Word remoteness, 137
Würtzburg School, 117

Members of the Hixon Symposium



Left to right: (seated) Halstead, Lashley, Klüver, Köhler, Lorente de Nó; (standing) Brosin, Jeffress, Weiss, Lindsley, von Neumann, Nielsen, Gerard, Liddell. Dr. McCulloch was unable to be present when this picture was taken.

Form No. 3.

PSY, RES.L-1

Bureau of Educational & Psychological Research Library.

The book is to be returned within the date stamped last.

29 DEC 1961		

WBGP-59/60-5119C-5M

150.1943
JEE
Form No. 4

BOOK C

Coll. No 150.1943
Author Jeffres
Title Cerebral M

Date.	Issued
29 DEC 1961	56

150.1943
JEE

The ORGANIZATION of BEHAVIOR
A Neuropsychological Theory

By D. O. Hebb, *McGill University.* Dr. Hebb combines present-day knowledge of psychology and physiology into a unique theory of thought and emotion—an attempt to explain what goes on in the human brain between the arrival of an excitation at a sensory projection area and its departure from the motor area of the cortex. In the first five chapters, Dr. Hebb discusses the problems involved in studying behavior and explains the theory he developed to solve these problems. From Chapter 6 on, the theory is applied to learning, volition, emotion, hunger, and similar factors in behavior. *The first volume of the* Wiley Publications in the Mental Health Sciences. *1949. 335 pages. $4.00.*

HANDBOOK of EXPERIMENTAL PSYCHOLOGY

Edited by S. S. Stevens, *Harvard University.* 34 leading scientists give authoritative accounts of such fields of experimental psychology as neurophysiology, homeostasis, endocrine functions, measurement, psychophysics, sensory functions, learning, motivation, perception, selection, training, and many others. *Handbook of Experimental Psychology* is the definitive book in its field—the most complete coverage of the subject available in a single volume. The 36 chapters have been logically divided among six sections: physiological mechanisms, growth and development, motivation, learning and adjustment, sensory processes, and human performance. *1951. 1436 double-column pages. $15.00.*

CYBERNETICS
or Control and Communication in the Animal and the Machine

By Norbert Wiener, *The Massachusetts Institute of Technology.* Norbert Wiener's *Cybernetics* needs very little introduction even to casual students of control, communication, and behavior. One of the few speculative books on science that have appeared in the United States, *Cybernetics* has become "a book to own, to read, and reread." In discussions that are for the most part easy for the layman to follow, Dr. Wiener attempts to find the common elements in the functioning of automatic machines and of the human nervous system, and to develop a theory which will cover the entire field of control and communication in machines and in living organisms. A TECHNOLOGY PRESS PUBLICATION, M.I.T. *1948. 194 pages. $3.00.*

The TRANSMISSION of NERVE IMPULSES at NEUROEFFECTOR JUNCTIONS and PERIPHERAL SYNAPSES

By Arturo Rosenblueth, *Instituto Nacional de Cardiología de México.* This book presents in systematic form the knowledge of chemical transmission at the junctions of motor nerves with striated muscles and at the synapses in autonomic ganglia. It covers all the important advances made in the field since 1937 when the author and Dr. Walter B. Cannon published a monograph on the subject. Besides revealing the more recently discovered facts on this transmission, the book reevaluates the theories which the author and Dr. Cannon accepted and defended more than a decade ago. A TECHNOLOGY PRESS PUBLICATION, M.I.T. *1950. 325 pages. $6.00.*

JOHN WILEY & SONS, Inc., 440 Fourth Ave., New York 16, N. Y.

PRINTED IN U.S.A.